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STREPTOCOCCIC INFECTION OF THE LUNGS FROM THE PARANASAL SINUSES

AN EXPERIMENTAL STUDY

OLOF LARSELL, PH.D.

LYLE VEAZIE, M.A.

AND

RALPH A. FENTON, M.D.

PORTLAND, ORE.

The relation of certain types of pulmonary infection to drainage from the paranasal sinuses has been emphasized by Mullin [1] in a series of papers. The various routes by which such drainage may reach the lungs have been discussed by Mullin,[1] Lawson [2] and others. More recently the results of experimental studies [3] have shown clearly that probably the most important pathway is a route combining the lymphatic and the blood vessels from the sinus membranes to the pulmonary capillary bed via the right side of the heart. These conclusions were reached as a result of injecting trypan blue into the sinuses and related regions under conditions which precluded the tracheal pathway to the lungs. Later, at various intervals, the trypan blue was found in phagocytes in the lungs and at various points along the route indicated.

On the basis of these results, experiments were undertaken to determine if cultures of streptococci introduced into the paranasal

From the Departments of Anatomy and Otolaryngology of the University of Oregon Medical School.

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sinuses and the lymph nodes receiving drainage from these sinuses follow the same route in animals. The present report deals with these experiments.

MATERIAL AND METHODS

Three series of animals, including cats and rabbits, were inoculated with cultures of streptococci. As a rule, inoculation was made into the frontal sinuses or into the paralaryngeal lymph nodes which drain these sinuses. The maxillary sinuses were inoculated in two rabbits. The animals were killed after periods of from one to one hundred and forty-four hours. The paralaryngeal lymph nodes, the lungs, parts of the liver and parts of the spleen were removed with aseptic precautions, and pieces were taken from each for bacteriologic culture. In some cases mediastinal lymph nodes were removed. Other pieces of the various organs were fixed in formaldehyde-Zenker solution for histologic study after being stained by methods calculated to reveal the bacteria in the tissues, of which we found the MacCallum modification of Goodpasture's basic fuchsin and crystal violet stain the most useful. Bacteriologic cultures of blood from the right side of the heart were made as a routine.

At autopsy the lungs of most of the animals showed more or less evidence of infection, ranging from small spots to considerable consolidation. These gross signs were not regarded as necessarily due to the experimental inoculations, since cats and rabbits frequently show pulmonary lesions without inoculation. Only when the beta streptococcus was recovered by bacteriologic methods or morphologically similar forms were seen in stained sections was the result regarded as positive. There is no attempt to indicate in the table the relative number of organisms, although this varied in different animals. The results are regarded as entirely qualitative and as demonstrating anatomic pathways rather than the degree of infection or the reactions of the tissues. The histologic procedure served to determine whether the organisms shown by bacteriologic culture were present in the bronchi or in the parenchyma of the lungs. They were virtually absent from the bronchial passages but were found in the parenchyma and the lymphatic vessels, as more fully described. The tracheobronchial route of entry cannot be as rigidly excluded for bacteria as it was for trypan blue in the earlier experiments, because the insertion of a tracheal cannula to eliminate material from the upper part of the respiratory region in itself favors bacterial contamination of the air passages. Cannulas, therefore, were not used.

In spite of precautions against dripping of inoculated material from the frontal sinuses, especially into the nasal cavity and thence into the trachea, this possible pathway to the lungs must be considered. The precautions consisted of injury to the sinus mucosa in the region of the ostia in a manner designed to produce swelling of the membranes and thus closure of the ostia. The position of the sinus openings into the nasal cavity and the posture of the head in the animals used were such as to prevent dripping into the nasopharynx or the trachea. It is believed on these grounds, as well as on the ground of histologic study of the bronchi and the lungs, that the tracheal route is negligible in these experiments. In the animals in which inoculation was made directly into the paralaryngeal lymph nodes the possibility of the tracheal route was entirely eliminated.

The first group of animals, designated series A, consisted of five cats. These were inoculated with a suspension in saline solution of a twenty-four hour blood agar culture of a feebly hemolytic beta streptococcus isolated from the frontal sinus of a human patient. Between 500,000 and 1,000,000 organisms were introduced into each of the animals of this series by the routes indicated in the table and were allowed to act for the periods indicated in the table.

Results of Experimental Inoculation of Cultures of Streptococcus into the Paranasal Sinuses and the Paralaryngeal Lymph Nodes, with Effects on Various Other Organs

Animal Number	Route of Inoculation	Number of Streptococci (Approximate)	Interval Between Inoculation and Death of Animal, Hours	Result of Culture						Histologic Observations				Comment
				Paralaryngeal Lymph Node	Lung	Liver	Spleen	Cardiac Blood	Mediastinal Lymph Node	Paralaryngeal Lymph Node	Lung	Liver	Spleen	
Cats														
A-2	Post. wall phar....	0.5 mil.	3*	N.C.†	—	—	—	—	—	+	Con.‡	Con.	Con.	+ Found dead in cage
A-4	Frontal sinus.....	1 mil.	4*	N.C.	—	—	—	—	—	..	..	..	..	Found dead in cage; no histologic study
A-3	Frontal sinus.....	1 mil.	96	N.C.	—	—	—	—	—	+	—	..	..	Lymph nodes enlarged; lymph nodules in lung enlarged
A-1	Post. wall phar....	0.5 mil.	120	N.C.	—	—	—	—	—	..	+	..	..	Lymph nodes enlarged
A-5	Frontal sinus.....	1 mil.	144	N.C.	—	—	—	—	—	..	+	..	..	Lower lobes of lungs consolidated; lymph nodes, spleen and liver enlarged
37-C-6	Region of L. par. L. N.	1 bil.	1	—	—	—	—	—	—	..	+	..	+	Killed one hour after inoculation
37-C-2	R. and L. par. L. N.	2 bil.	3½*	N.C.	—	—	—	—	—	..	+	+	..	Found dead in cage 3½ hours after inoculation
37-C-8	R. and L. par. L. N.	1 bil.	3	N.C.	—	—	—	—	N.C.	+	+	+	+	Lungs showed large mottled areas
37-C-5	R. and L. par. L. N.	2 bil.	5	+	+	+	+	2 P CC.	—	+	+	+	+	
37-C-7	Region of par. L. N.	2 bil.	5	N.C.	—	+	—	1,200 P CC.	+	+	+	+	?	Lungs mottled
37-C-1	R. and L. par. L. N.	2 bil.	24	+	+	+	+	+	+	+	+	..	+	Found dead in cage; regional lymph nodes greatly enlarged; lungs consolidated
37-C-4	L. par. L. N.....	0.4 bil.	48	+	+	—	—	—	+	+	..	..		Regional lymph nodes enlarged; spleen dark; lungs normal grossly
37-C-3	L. par. L. N.....	40 mil.	72	+	+	+	+	—	—	+	+	+	+	Regional lymph nodes enlarged; spleen dark; lungs normal grossly
37-C-10	L. fr. sinus.....	1.5 bil.	2½	—	+	—	—	—	..	+	?	..	+	Died 2½ hours after inoculation; spleen greatly enlarged
37-C-11	L. fr. sinus.....	6 bil.	20*	..	—	..	—	—	N.C.	..	..	..	..	No gross pathologic condition; apparently died from anesthetic
37-C-9	L. fr. sinus.....	6 bil.	48	—	..	..	..	—	—	+	+	+	+	Spleen large and dark; regional lymph nodes large
37-C-12	L. fr. sinus.....	4 bil.	..	.	..	..	..	..	..	..	..	..	..	Died from anesthetic
Rabbits														
37-R-4	R. and L. par. L. N.	2 bil.	1	+	—	—	—	—	N.C.	+	+	?	..	Died one hour after inoculation (anesthetic) regional lymph nodes filled with fluid
37-R-1	Region of R. and L. par. L. N.	2 bil.	2½	N.C.	—	—	N.C.	—	N.C.	..	+	..	..	Died about 2 hours after inoculation; lymph nodules in lung enlarged
37-R-3	Region of R. and L. par. L. N.	2 bil.	3	N.C.	Con.	+	+	400 + P CC.	..	+	+	+	+	Petechial spots in lung
37-R-2	Region of R. and L. par. L. N.	2 bil.	7	N.C.	+	+	+	4 P CC. +	N.C.	+	+	+	+	
37-R-6	R. max. sinus.....	3 bil.	24	—	+	+	+	—	..	+	+	..	..	Regional lymph nodes enlarged
37-R-5	R. max. sinus.....	3 bil.	48	Con.	Con.	—	—	—	..	..	..	..	..	Paralaryngeal nodes enlarged; lungs mottled at margins

* Approximate. † Not cultured. ‡ Contaminated.

It was soon apparent that the paralaryngeal lymph nodes filtered out the bacteria pretty thoroughly when inoculation was made into the region of the paranasal sinuses. In series A, therefore, in which relatively small doses were used, the results of bacteriologic study of the organs were negative, as indicated in the table.

In series 37-C, consisting of twelve cats, and in series 37-R, consisting of six rabbits, a new culture of a beta streptococcus was used. The concentration was made greater than that used in series A by suspension of the sediment from 25 cc. of a twenty-four hour Hartley broth [4] culture in 1 cc. solution of sodium chloride and injecting this mass into the sinuses or the lymph nodes. This procedure gave individual doses of from 40,000,000 to 6,000,000,000 organisms, as shown in the table. The Hartley broth cultures were made as for series A, quantitative blood cultures being made in blood agar plates. Pieces of the lymph nodes, the lung, the liver and the spleen were fixed for histologic study. Pentobarbital sodium anesthesia was used during all the operative procedures, the effect of ether on the respiratory tract thus being avoided.

OBSERVATIONS

Hartley broth cultures of the lung, the liver, the spleen, the cardiac blood and the mediastinal lymph nodes gave negative results in series A, as already indicated. Histologically, two of the animals showed streptococci in phagocytes within the paralaryngeal lymph nodes, and two others showed streptococci in the lungs. In series 37-C and 37-R, which received massive doses of the organism, the paralaryngeal lymph nodes were apparently unable to cope with the invasion, and many of the organisms filtered through them into the efferent lymphatic vessels and finally into the blood stream. Culture of the lungs, the liver and the spleen of these animals gave positive results as early as five hours (in one rabbit three hours) after inoculation. Tissue from most of the animals, as indicated in the chart, showed streptococci histologically, even when the results of culture were negative. This raises the question of viability of the streptococcus after its ingestion by phagocytes. Possibly the ingested organisms shown in stained sections were not viable, but of this we have no evidence save that culture of the organs in which they were found did not give positive results. Another possibility must be recognized, namely, that the pieces of the various organs cultured may not have been reached by the organisms. In either case it is clear, as would be expected, that the inoculation of large numbers of bacteria into either the sinuses or the paralaryngeal lymph nodes is more effective in causing viable bacteria to reach the lungs and other organs than that of small numbers. This suggests that positive results obtained by bacteriologic methods may have been due to the presence of free streptococci within the organs cultured. Figure 1 *A* shows that these were present in the pulmonary parenchyma of some of the animals, both as individual organisms and in small masses, some of which suggested small thrombi, apparently in the capillaries.

4. Hartley broth is made of a pancreatic digest of beef muscle.

Streptococcus-laden phagocytes were found in the lymph nodules along the bronchial walls within the lungs, in the bronchial lymphatic vessels (fig. 1 *B*) and in small numbers in the lymphatic vessels of the pleura. They are seen thus to appear in the same anatomic locations

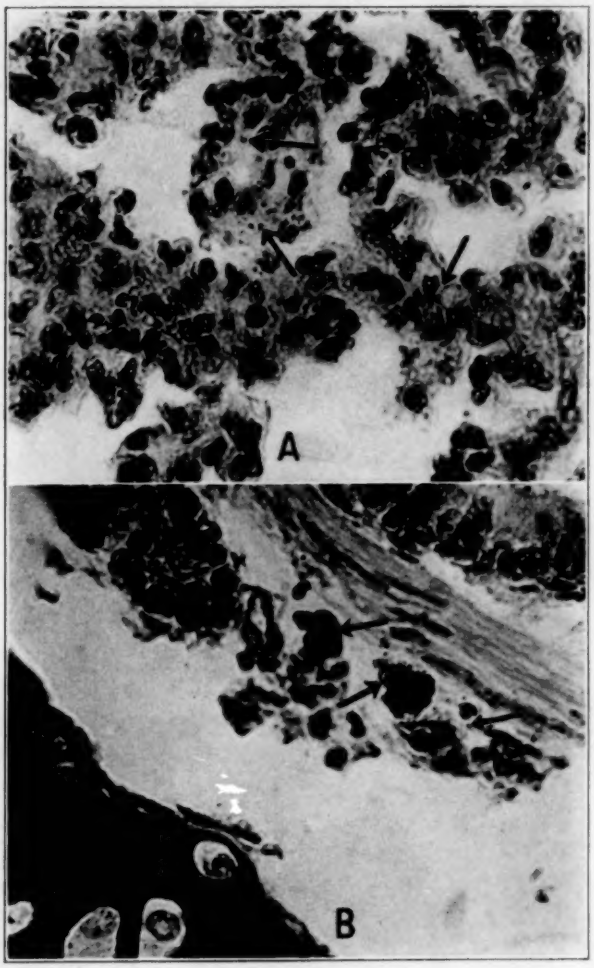

Fig. 1.—*A*, streptococci in the parenchyma of the lung of a cat whose frontal sinus had been inoculated with a concentrated culture; *B*, phagocytes laden with streptococci in the bronchial wall of the same animal.

within the lung as did the trypan blue-laden phagocytes observed in the earlier experiments. The bronchial lymph nodules, usually small in the cat, were enlarged, especially in the animals kept alive for some time after inoculation. There was also an increase of the normally relatively large bronchial lymph nodules of the rabbit's lung.

Sections of the paralaryngeal lymph nodes, the liver and the spleen, fixed between one and twenty-four hours after inoculation, showed free streptococci in the tissues (fig. 2 *A*), as well as ingested bacteria

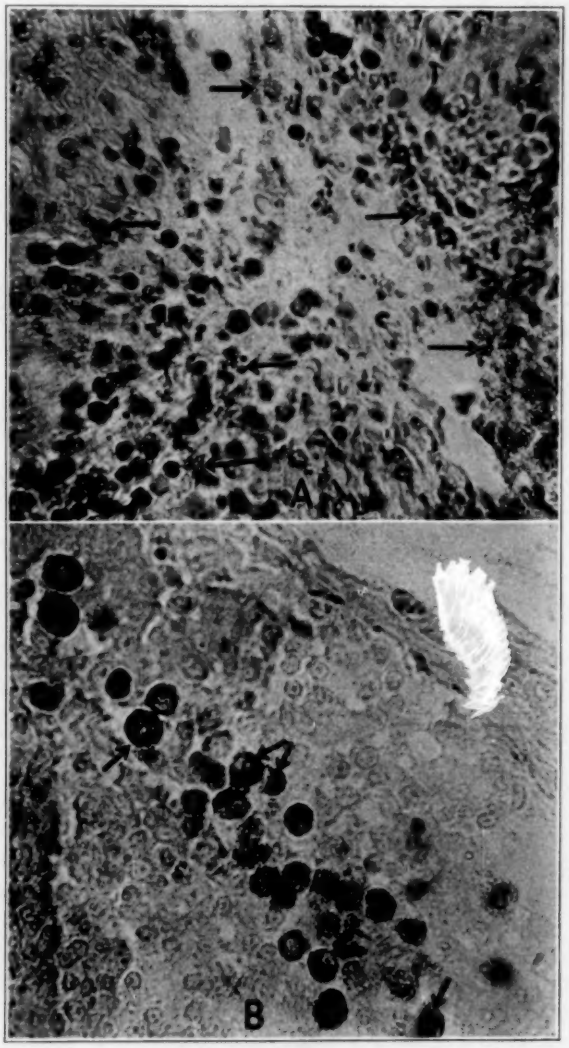

Fig. 2.—*A*, paralaryngeal lymph node of a rabbit with masses of free streptococci in the sinuses three hours after inoculation into the node; *B*, streptococcus-laden phagocytes in the peripheral sinus of the paralaryngeal lymph node after inoculation of a concentrated culture into the frontal paranasal sinus of a cat.

(figs. 2 *B* and 3). The number varied with the organ and the animal, as well as with the period after inoculation.

The series of animals is too small to permit fixed conclusions regarding the time factor in the clearing of the inoculated bacteria from the blood stream. The results clearly show, however, that the blood stream is an important link in the dissemination of the organisms from the sinus region to the lungs, the liver and the spleen, and presumably to other parts of the body, not examined in this study. Under the conditions of the experiment the most obvious route from the sinus membranes was that via the lymphatic vessels to the paralaryngeal lymph nodes, thence through the paratracheal lymph vessels to the great veins and on into the right side of the heart, from which blood was taken for culture.

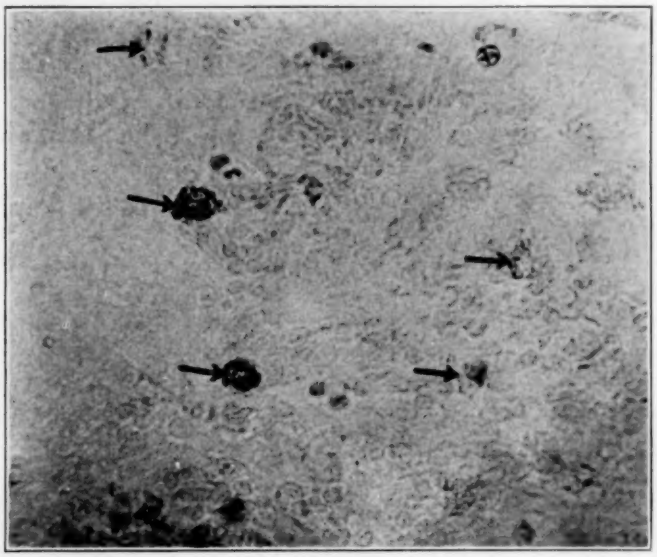

Fig. 3.—Streptococcus-laden phagocytes in the deep sinuses of a paralaryngeal lymph node of a cat after inoculation into the lymph node.

It would be of interest to compare cultures of blood from the right side of the heart with similar cultures of blood from the left side, with reference to number of organisms and other features, but this has not been done. The results of both bacteriologic methods and histologic study of the liver and the spleen indicate that the lungs do not constitute so effective a filter for streptococci as they do for trypan blue, since relatively little of the latter substance could be found in the liver and the spleen in the earlier experiments. On the basis of cultures, it appears that the blood stream is pretty well cleared of organisms under the conditions of these experiments in about twenty-four hours, since for only one animal of the twenty-three used were the results of blood culture positive as late as twenty-four hours after inoculation.

SUMMARY AND CONCLUSIONS

Viable streptococci, as shown by bacteriologic methods and checked as to the pathway followed by histologic control, can reach the lungs from the paranasal sinuses and the related regional lymph nodes.

The number of organisms reaching the lung is much less when the sinuses are inoculated than when the bacteria are introduced directly into the paralaryngeal lymph nodes, which normally serve as the filters for the lymphatic stream from the sinus region.

The fact that streptococci reach the lungs, the liver and the spleen from the paralaryngeal lymph nodes when either sinuses or lymph nodes are inoculated indicates that the anatomic path must be via the paratracheal lymphatic vessels to the great veins and thence into the right side of the heart and into the pulmonary capillary bud. From this point they may reach the pulmonary tissues or be carried by the blood stream to other parts of the body. Most of the organisms that pass through the lungs are apparently filtered out of the blood stream by the spleen and by the Kupffer cells of the liver.

HEARING OF SPEECH

DOUGLAS MACFARLAN, M.D.
PHILADELPHIA

The importance of the hearing of speech and a plea for the serious consideration of it in the study of otology can be accentuated by the following axiomatic statements:

Language—that coin in the realm of communication—can be only partially learned when there is poor hearing of speech.

Education is slow, difficult, partial or incomplete without hearing of speech.

Economically, a person is usually greatly handicapped by inability to hear speech well.

Socially, the hard of hearing and the deaf are often seriously maladjusted.

As a consequence of the appreciation of these facts, there is a deservedly growing interest in speech hearing among educators, sociologists and psychologists, as well as among otologists. More than any one else, the deafened man himself insists on the seriousness of his situation.

Otologists in the past have focused their attention largely on the patient's efficiency in hearing pitches within the hearing range, and they have with difficulty tried to get from this a conception of the loss of hearing for speech. A plan has been devised to add together the losses for the frequencies and divide them by a factor which will give an approximation of the percentage loss of hearing. But speech hearing is such a subtle thing, and single tone hearing is so different from it, that the comparison is almost useless. In my own examinations over many years, I can put no confidence in this method of inferring what speech hearing is.

There is no better way of testing speech hearing than by the use of speech itself as a stimulus. This fact is gaining ground with all those who by experience familiarize themselves with the subject. The use of the phonograph has made it possible for the voice to be reproduced at roughly an even level, and one is sure that repetition of the testings at various dates gives comparatively identical testing conditions.

There are, however, certain incorrigible factors of variability which must be understood and recognized. They signify that the test is not faultless; no test is. These variables may be stated categorically:

No two speech sounds are alike in frequency characteristics.

No two words are acoustically equally well heard.

Accent and inflection are factors.

Many words are not equally familiar.

Vowel sounds are recognized more easily than consonant sounds, but consonants are of double the importance of vowels in the proper interpretation of the word.

Attention and fatigue are large factors in testings.

As in all hearing testings, dampening by outside noise or by fleeting sounds that may interrupt at critical hearing moments is most important.

All these factors will make it impossible ever to say that testing of speech hearing with speech is a perfect test; yet there is nothing better. Many of these variables are impossible to overcome, but many can be stabilized.

In order to minimize the factor of the varying acoustic value of test words, it is advisable to use many, not few, words. Credit is given the patient for the level at which he hears the majority of words correctly. The selection of test words should be made from Thorndyke's table of words in most common use in the English language, the monosyllables being chosen. Thus, the factor of familiarity is reduced to a minimum.

GRADING THE LOSS

Fletcher has stated that there is a span of 100 decibels between barely audible speech and speech so loud that it reaches the auditory-tactile sense. Thus, any loss in decibels is equivalent to percentage hearing loss. I feel, however, that it is not yet time to say what percentage loss of hearing is and that one will be on more sure ground if one reports the decibel loss.

When Snellen introduced the visual test type, he selected an angle subtending 5 degrees as a unit of size. Illumination was to be merely good light. Yet the matter of what was good or normal vision was left in abeyance until experience developed a rating to be considered normal. Hearing acuity and visual acuity are both known to vary with age. Such matters as acuity are the concern of physiologists and of persons daily going through the experience of frequent testings. It would seem a mistake, then, to adopt the term "percentage" too early. The decibel, a physical unit, can express the loss in hearing, and time and familiarity will tell where the normal lies.

HEARING AIDS

Advantages and Disadvantages.—Many practical problems are brought to otologists by deafened patients. One of the commonest is presented by the question "Shall I wear a hearing aid?" The question is many sided, but the problem may be resolved into factors that can be evaluated. Benjamin Franklin in considering a mooted point advised the use of two columns, headed "Why I Should" and "Why I Should Not." The method in this case seems to offer help. The otologist certainly should first establish in his mind a level of hearing loss at which he considers it advisable to recommend the use of a hearing aid. This

implies, of course, testing of speech hearing to determine the decibel loss. The patient's occupation and necessities are important considerations as well. Familiarity, convenience and conspicuousness can all be adjusted. Actual trial of an aid under testing conditions by the otologist examiner is essential. A convenient interpretation test is used.

One important thing to remember is that the hearing aids usually amplify noise as well as speech, and today the world is full of noise. Noise interferes with or dampens speech hearing. Speech comes through at cycles ranging roughly from 300 to 3000. Most common noises are below 500 cycles. Speech hearing is most affected by the hearing in the zone 1000 to 2000. The low tones are not very important, yet many aids amplify them.

Phonograph Testing to Facilitate Selection.—There is a convenient method of testing a patient's need of hearing low tones and from this information selecting a proper aid. Using a phonograph with a control capable of varying the speed of the disk, set the attenuator slightly above the patient's decided loss. Use a constant level speech record with the disk revolving at the standard rate, 78 revolutions per minute. Take a record of the patient's ability to interpret under these conditions, using as an index the percentage of correct answers. Now, leaving the level of sound intensity the same, increase the speed of the record. The low tones fade, and the high tones become conspicuous. Make another test of the percentage of ability to interpret. It is usually found that the patient makes a better showing with the faster record. The test has been of much value to otologists in advising patients as to the selection of an aid.

IMPROVED APPARATUS FOR RECORDING SPEECH

Within the year there have appeared the crystal receiver and the crystal phonograph pick-up, which have revolutionized the quality of speech obtainable from a record. The new apparatus avoids the peaking in the 1000 cycle zone that was such a factor in the older disk receivers and in the carbon microphones. Further, frequencies up to 7000 and 8000 cycles are transmissible with the newer apparatus. The amplification of speech across its frequency range is now possible electrically for the first time without distortion.

CONCLUSION

Testing of speech hearing with phonographic speech is an indispensable adjunct to the study of deafness by otologists. It discovers the degree of disability to the hearing of that which the patient is most concerned in hearing. A knowledge of the subject opens up an opportunity to serve the deafened patient.

1805 Chestnut Street.

COD LIVER OIL FOR THE LOCAL TREATMENT OF TUBERCULOUS PHARYNGITIS AND LARYNGITIS

ANDREW L. BANYAI, M.D.
WAUWATOSA, WIS.

A careful search of the *Quarterly Cumulative Index Medicus* since 1926 failed to reveal any publication in the literature dealing with the topical application of cod liver oil in treatment of tuberculous pharyngitis and laryngitis. For this reason it seems to be justifiable to present the experience with this remedy that I gained during the past one and a half years.

Cod liver oil was employed as a popular household remedy in the Scandinavian countries for centuries. It not only was used internally for a great many diseases but was known to have a healing effect when applied externally to cut wounds, abrasions and ulcers of the skin. Its systematic application in hospitals was introduced in Scotland during the second half of the eighteenth century, and it attained a widespread application in medical practice in Europe from 1840 on.

Experience showed that it is of value in tuberculosis. The factors responsible for its therapeutic effect were not known, however, for a long time. Some thought that it is useful only because of its easy absorbability from the intestinal tract and because of its easy digestibility. Others attributed its beneficial influence to an increased resistance against tuberculosis that could be produced experimentally in animals kept on a high fat diet. Empirically it was shown also in some instances that the fever of tuberculous patients can be lowered by the use of cod liver oil.

The mysticism that surrounded the underlying cause of the therapeutic action of cod liver oil in rickets, tuberculosis and keratomalacia and in the treatment of infected wounds and ulcers began to be disposed of and replaced by scientific facts from the beginning of the twentieth century. The names of McCollum and Davis, Osborne and Mendel are associated with the discovery of vitamin A, and the English physiologist Mellanby is credited with the discovery of vitamin D. It is generally accepted today that the large amounts of these two vitamins are responsible for the curative action of cod liver oil. The probable importance of its iodine content and the influence of the latter on some of the toxic manifestations of tuberculosis (fever and night

From the Muirdale Sanatorium.

sweats) are not sufficiently appreciated. Holmes and Remington [1] found that American cod liver oil contains more iodine than do most ordinary foods, the average being nine parts per million, and that 10 cc. of high grade medicinal cod liver oil would cover the daily iodine requirement, which is 50 to 100 micrograms. According to the Eleventh Revision of the United States Pharmacopeia, standard medicinal cod liver oil must have a vitamin A potency of not less than 600 U. S. P. units per gram and a vitamin D potency of not less than 85 U. S. P. units per gram.

In a search for an understanding of the direct action of cod liver oil on the tubercle bacillus, Campbell and Kiefer [2] cultured tubercle bacilli on a potato medium that was soaked with cod liver oil. After five weeks' growth the bacteria presented marked degenerative changes. When stained by the Gabbet method the bacilli appeared under the microscope smaller, beaded or dumb-bell shaped. Many coccus-shaped single acid-fast organisms were seen. With the granule stain the red-staining protoplasm was not evident. These investigators attributed the inhibitory effect to some unknown constituent of the cod liver oil.

I began the topical application of cod liver oil in 1934. My first report on this subject [3] was based on the observation of forty-seven cases. Of these there were three of lupus vulgaris, one of scrofuloderma, one of multiple subcutaneous tuberculous abscess, six of tuberculous lymphadenitis, one of so-called primary tuberculosis of the muscle, three of tuberculous empyema, one of sinus disease following tuberculous epididymitis, four of ischiorectal fistula, two of tuberculous ulcer of the pharynx and twenty-five of tuberculous laryngitis.

I received encouragement for the local use of cod liver oil from the works of Loehr,[4] who employed it in a large number of nontuberculous conditions with success. He reported that cod liver oil exerts a striking inhibitory effect on the bacterial flora of wounds. He found also that bacteria ordinarily encountered in infected wounds, such as streptococci, staphylococci and Bacillus coli, perish when introduced into cod liver oil.

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To test the actual healing power of cod liver oil in tuberculosis, a patient with lupus vulgaris was selected for treatment. The disease was of six years' duration. All previous therapeutic efforts had failed, although the patient was under the care of competent dermatologists. Small pieces of gauze soaked in cod liver oil were applied to the involved areas twice a day. A favorable change was noticeable shortly after the beginning of the treatment. A rich granulation tissue gradually filled in the deeply ulcerated areas. This was followed by prompt epithelization. Complete healing resulted in eight months.

In view of the fact that the lupus involved practically the entire face and the entire extent of the skin of the left arm, the results must be considered rather extraordinary.

Subsequent favorable responses were seen in other tuberculous lesions. Encouraged by these, I made an attempt to treat tuberculous pharyngitis and laryngitis by a cod liver oil spray. Ordinary medicinal cod liver oil was used. Sterilization of the oil is not necessary; it is free from bacteria. The following case reports are typical illustrations.

L. L., a woman 20 years old, had far advanced pulmonary tuberculosis, tuberculosis of the tonsils and peritonsillar structures and tuberculous laryngitis. She stated that the condition of her throat developed two months before her admission to the sanatorium. She complained of sore throat and dysphagia. Both anterior and posterior pillars and both tonsils were markedly congested; the right tonsil and the right anterior pillar were ulcerated. The same involvement was present on the left side, although it was not extensive. The treatment consisted of cleansing the throat with compound solution of sodium borate and spraying it with cod liver oil three times a day. In three weeks the ulcers disappeared, the margins of the pillars were smooth and the congestion of these structures cleared entirely, except in the posterior part of the right tonsillar fossa; this region, however, was normal four weeks later. The soreness of the throat and the dysphagia disappeared.

F. A., a man 37 years old, had far advanced pulmonary tuberculosis and tuberculosis of the pharynx. He complained of marked dysphagia and sore throat. Because of the pain in the throat, he was using a cocaine spray several times a day prior to the beginning of the treatment with cod liver oil. There was an ulcer the size of a bean in the left tonsillar fossa. The ulceration extended to the posterior pillar. An ulcer of approximately the same size was visible on the posterior pharyngeal wall to the right of the uvula. The remainder of the pharyngeal structures were moderately congested. After one month's treatment with a cod liver oil spray to the pharynx, the ulcers healed, except a split-pea-sized area of the left tonsillar fossa that still showed some membrane. At this time the treatment had to be discontinued because the patient acquired tuberculous meningitis that caused his death. Observations made during the course of treatment showed gradual clearing of the ulcerated, necrosed areas and disappearance of the precipitate that covered the ulcers.

In my third case of tuberculous pharyngitis (A. E., a woman 23 years old) tuberculous meningitis terminated the treatment also. When the patient was first examined, she complained of intense sore throat and dysphagia. The posterior pharyngeal wall, the uvula and the anterior pillars showed extensive "moth-eaten" ulcerations. The midsection of the right anterior pillar revealed a marked loss of tissue. The tonsils had a chewed-out appearance. All ulcerated and necrotic

areas were covered with white exudate. The entire throat was markedly congested. Multiple tuberculous ulcerations were present at the root of the tongue. This patient was in the preterminal stage at the time of her admission. Still, in spite of her low vitality, clearing and improvement in the ulcerated areas were detectable during the first two weeks of treatment with the cod liver oil spray.

Complete healing followed the topical application of cod liver oil in a case of tuberculous ulceration of the pharynx. B. S., a white woman 19 years old, had far advanced pulmonary tuberculosis and tuberculous laryngitis and pharyngitis.

In another case the treatment was of short duration because of the death of the patient (R. H.).

One patient, J. J., stated that the application of the oil increased the soreness of the throat. For this reason the treatment was discontinued. He died three days later. The pharyngeal findings were a large tuberculous ulcer destroying the left tonsil and the left anterior pillar and several pinhead-sized ulcers on the left side of the soft palate.

The laryngeal application of cod liver oil was begun with considerable hesitation because of the fact that several reports of so-called oil aspiration pneumonia have appeared in the past few years. Laughten [5] demonstrated clinically and also in experiments on animals that oil finds its way into the alveoli of the lung and produces pneumonia when administered in sufficient quantities in the nose and throat. Pinkerton [6] and Baumgartner and Murray Angevine [7] reported cases of oil pneumonia caused by cod liver oil. The patients, however, were very young children, whose lungs do not have great power of expulsion.

All patients treated by me for laryngeal tuberculosis were adults. All had considerable productive cough. I thought that if some of the oil sprayed into the larynx reached the lung it would be removed effectively by the cough. In ninety-one patients who were under observation from two months to one and a half years oil pneumonia did not occur. It may be stated with assurance, therefore, that the prolonged use of a cod liver oil spray to the larynx is not likely to cause oil pneumonia in adults.

The treatment of the larynx consisted of spraying it with cod liver oil by means of an ordinary atomizer three times a day. The spray was always given after meals for avoiding possible anorexia caused by the taste of the oil. The oil must be warmed prior to its application for two reasons: (1) Heating diminishes its viscosity, and (2) the diseased mucous membrane tolerates warm oil better than cold oil. The nurse or attendant, instructed in the technic, holds the tip of the atomizer

5. Laughten, A. F.: Studies on Pneumonia Following Nasopharyngeal Injection of Oil, Am. J. Path. **1**:407, 1925.

6. Pinkerton, H.: Oils and Fats: Their Entrance into and Fate in the Lungs of Infants and Children; a Clinical and Pathologic Report, Am. J. Dis. Child. **33**:259 (Feb.) 1927.

7. Baumgartner, L., and Angevine, D. M.: Lipoid Pneumonia and Conditions That May Favor Its Occurrence, Am. J. M. Sc. **192**:252, 1936.

just beyond the root of the tongue directed downward without touching the pharyngeal structures, while the patient is told to hold the tongue between the thumb and the index finger. From ten to twelve compressions of the bulb of the atomizer deliver a sufficient amount of oil into the accessible parts of the larynx.

There were only a few patients who did not tolerate cod liver oil because of its fishy taste. Two stated that their throats became sore and irritated by the oil. Two were unable to take it because the spray caused nausea and vomiting. One patient related that the oil caused irritation in the throat and provoked a coughing spell.

Since the institution of treatment with cod liver oil at the Muirdale Sanatorium all patients whose condition was diagnosed as laryngeal tuberculosis were treated by this method. All these patients had active pulmonary tuberculosis. Some had other extrapulmonary complications. The seriousness of their condition is best illustrated by the fact that of 120 patients with laryngeal tuberculosis 37, or 30.8 per cent, died during the course of the treatment, the majority shortly after the beginning.

The laryngeal findings were those characteristic of tuberculosis associated with a chronic pulmonary process. Several patients had a combination of the different manifestations of the disease, such as infiltration, ulceration, interarytenoid vegetative granulation and edema. The evaluation of the effect of cod liver oil on laryngeal tuberculosis is rather difficult for several reasons. Tuberculous laryngitis may show spontaneous healing in its early stages. Its course may parallel that of the pulmonary disease. When the general condition of the patient, his immunity and his defense are poor, the chances for improvement in serious laryngeal tuberculosis are slight.

In patients who responded favorably to the treatment the relief from disagreeable symptoms was rather prompt. Several of the patients with ulcer who used a cocaine spray or ethyl aminobenzoate tablets before the treatment were able to discontinue these shortly after the use of cod liver oil began. The disappearance of dryness, tickling and burning sensations from the throat and the cessation of pain that was localized to the larynx or radiated to the ear were frequently observed. With the elimination of the pain, dysphagia was relieved and the patient's nutrition and general well-being improved. It was also noted that the cough diminished and expectoration became easier. With the diminished cough and lessened expectoration, more pulmonary and general rest and better, more quiet sleep were insured for the patient. The gradual disappearance of hoarseness and the restoration of normal voice were impressive for the patients as well as for the attending staff. Such favorable changes were followed by an improved, more optimistic mental attitude on the part of the patient.

The change in the laryngeal findings was striking as the treatment progressed. Ulcers showed rapid epithelization and healing, which were comparable with the results observed in cases of pharyngeal tuberculosis, provided the treatment was carried out consistently and was well tolerated by the patient. Similarly, a good therapeutic response was observed in patients with various degrees of vegetative granulation. Other manifestations of the disease also cleared up satisfactorily. I had the least success in cases in which there was marked edema. Utter failure of the treatment in several of these cases was discouraging.

The analysis of the results in ninety-one patients for whom the treatment was continued from two months to one and a half years revealed that twenty-four, or 26.3 per cent, remained unimproved. Of these, eight died during the course of the treatment; forty-two, or 46.2 per cent, improved subjectively and objectively, and 25, or 27.5 per cent, healed. In the majority of the cases in which healing occurred, it was established before the patient recovered from the pulmonary tuberculosis; therefore, it is reasonable to assume that the healing was accomplished, aided or accelerated by the topical application of cod liver oil.

A brief mention of two more points may be of some interest. After having convinced myself of the therapeutic value of the plain cod liver oil, I have been prescribing it recently with 1 per cent menthol and camphor for some of the patients who had tried plain cod liver oil but were unable to take it. An attempt was made to promote the penetration of the oil into the tissues by preparing and spraying a 50 per cent emulsion into the larynx. The emulsion proved to be useless for internal application because of its extremely disagreeable taste.

CONCLUSIONS

A report on the topical application of cod liver oil in one hundred and twenty-six cases is presented. Of these, ninety-one cases of laryngeal and six of pharyngeal tuberculosis were analyzed. Thirty-seven patients with laryngeal tuberculosis died, twenty-nine in less than two months; their cases were not analyzed.

Cod liver oil can be used with safety in tuberculous pharyngitis and laryngitis.

Pharyngeal and laryngeal ulcers showed rapid epithelization and healing. A favorable therapeutic response was seen in cases in which there was localized infiltration or vegetative granulation. Laryngeal tuberculosis with marked edema is rather resistant to this treatment.

Of ninety-one patients with laryngeal tuberculosis, whose treatment lasted from two months to one and a half years, twenty-four, or 26.3 per cent, remained unimproved, forty-two, or 46.2 per cent, improved

subjectively and objectively and twenty-five, or 27.5 per cent, healed. In the majority of the cases in which healing occurred, it was established before the patient recovered from the pulmonary tuberculosis.

The evidences of symptomatic relief, such as elimination of pain, dysphagia and irritation in the throat, diminution of the cough, and easier expectoration, were promptly noticeable. Restoration of the normal voice, better sleep and improvement in the patient's general well-being accompanied the objective manifestations of healing.

TREATMENT OF RECURRENT VERTIGO (MÉNIÈRE'S SYNDROME) BY SUBTEMPORAL DESTRUCTION OF THE LABYRINTH

TRACY J. PUTNAM, M.D.
BOSTON

The syndrome of chronic recurrent explosive attacks of vertigo so severe as to incapacitate the patient and render life miserable and ordinarily unrelievable by sedatives is one which is remembered with distress by all physicians who have encountered it. Its etiology is varied.[1a] It may result from an acute or a chronic inflammation of the ear (chronic aural vertigo), from disease of the medulla, from fracture of the temporal bone and perhaps from a toxic condition. In many cases the cause is unknown, and these cases are often grouped together under the term Ménière's disease, although Ménière described the syndrome as arising from any one of a number of diseases. For therapeutic purposes, care should be taken to exclude from the classification all cases in which vertigo occurs during the course of an acute suppurative or irritative process in the ear, in which the symptom usually subsides with its cause, and also all cases of chronic mild constant dizziness, of which sufferers from cerebral arteriosclerosis and the late effects of head injury so often complain.

The phenomena which accompany chronic recurrent attacks of vertigo have been well described in recent publications.[1] In my experience the patient may or may not have a history of chronic disease of the ear. There is often a mixed type of deafness on both sides, with retraction of the drums and tinnitus. The labyrinths may be slightly irritable or slightly hypoactive to irrigation. Attacks are usually characterized by nausea, vomiting, falling and nystagmus. There is invariably a sensation of turning or of whistling during the attacks, but the patient may be entirely well between them.

From the Neurological Unit, Boston City Hospital, and the Department of Neurology, Harvard Medical School.

1. (*a*) Munro, D.: The Surgical Treatment of Certain Repeated Explosive Attacks of Vertigo Occurring in the Absence of Any Demonstrable Etiology: Ménière's Disease, New England J. Med. **216**:539 (April 1) 1937. (*b*) Dandy, W. E.: Ménière's Disease: Its Diagnosis and Method of Treatment, Arch. Surg. **16**: 1127 (June) 1928. (*c*) Coleman, C. C., and Lyerly, J. G.: Ménière's Disease: Diagnosis and Treatment, Arch. Neurol. & Psychiat. **29**:522 (March) 1933.

METHODS OF TREATMENT OF CHRONIC RECURRENT VERTIGO

Vertigo may be treated by medical or by surgical methods. The most successful nonoperative treatment is unquestionably the regimen proposed by Furstenberg,[2] consisting of a diet essentially free from sodium chloride plus the administration of large doses (up to 15 Gm.) of ammonium chloride daily. Why this treatment should be effective is not obvious, for it appears to make no impression on the sodium level of the blood and does not produce acidosis. It is, however, effective in many instances in relieving not only vertigo but, to a lesser extent, tinnitus, if there is any. I have found that ammonium chloride may be given in enteric-coated tablets, and my former associate Dr. R. Frank Slaughter has pointed out that the dose can sometimes be reduced and its effect increased by substituting ammonium bromide in part.[3]

The accepted surgical treatment in this country is section of the eighth nerve. This operation has given excellent results at the hands of Dandy,[1b] Coleman,[1c] Munro [1a] and others. McKenzie [4] has introduced the method of partial section of the nerve, which preserves hearing. Nearly 100 cases in which this method was used without a death, have been reported,[1a] but unreported deaths have occurred. In only 3 of the cases in the literature was the operation considered a failure. The degree of relief varies somewhat, however, and is not always complete. If concomitant tinnitus is present, it often persists after the operation. The procedure is not always as easy, rapid and safe as some accounts would lead one to believe.

An older operation, still extensively employed in England [5] and in France,[6] is destruction of the labyrinth, usually by injection of alcohol into the foramen rotundum or by exenteration of the canals. In either case the field is approached through the mastoid and the middle ear, which constitute an avenue not free from danger of hemorrhage or infection. Such operations have been necessarily followed by complete deafness on the side of the operation, which greatly limits their usefulness. They are successful, however, in relieving symptoms in most instances. They have been employed chiefly in cases in which symptoms followed a suppurative process; but Hautant has reported 13 cases of

2. Furstenberg, A. C.; Lashmet, F. H., and Lathrop, F.: Ménière's Symptom Complex: Medical Treatment, Ann. Otol., Rhin. & Laryng. **43**:1035, 1934.

3. Slaughter, R. F.: Personal communication to the author.

4. McKenzie, K. G.: Intracranial Division of the Vestibular Portion of the Auditory Nerve for Ménière's Disease, Canad. M. A. J. **34**:369, 1936.

5. Mollison, W. M.: Operative Treatment of Vertigo, Proc. Roy. Soc. Med. **28**:1597, 1935.

6. Hautant, A.: Indications et résultats éloignés du traitement chirurgical des vertiges auriculaires, Bull. Acad. de méd., Paris **111**:933, 1934.

"otosclerotic" vertigo without preceding otitis media (apparently corresponding to Ménière's disease as it is defined by most American surgeons) in which treatment by injection of alcohol into the labyrinth resulted in complete relief of the patient in 11 cases, partial relief in 4 and no change in 2. It should be remarked, however, that the case histories in most of the articles concerning operative destruction of the labyrinth are so inadequate that it is difficult to obtain a clear idea of what was done or of what the results were.

THEORETICAL BASIS OF THE PRESENT OPERATION

Physiologically, there can be no essential difference between destroying the labyrinth and cutting the fibers arising within it in the vestibular nerve provided destruction is complete in both cases. It is probably true that a more thorough destruction can be assured by section of the nerve than by reaching the end organ through the middle ear.

There is, however, another approach available. The semicircular canals and the beginning of the vestibular nerve are actually closest to the surface of the cranium at a point anterior to the lateral sinus in the temporal fossa. Anatomic studies make it clear that the superior canal can be reached easily and directly by making a small subtemporal decompression, elevating the dura as for a section of the trigeminal root and drilling the petrous portion anterior to the petrosal sinus (fig. 1). This method of approach keeps one well away from the large vessels, the cranial nerves and the brain stem, which are so uncomfortably close by in the posterior fossa. It permits a cleaner and more anatomic dissection than does approach through the mastoid, and gives a more certain exposure of the canals. Finally, it entails less danger of producing deafness than either of the other methods, since the middle and the internal ear are left intact and the auditory nerve is not even within the field. Indeed, the safety of the cochlea is actually a disadvantage in cases like the 2 reported here, in which tinnitus is such an annoying symptom that special efforts to destroy the hearing appear indicated. This operation was carried out five times in the 2 cases: three times on the same side in one instance, and bilaterally in the other.

REPORT OF CASES

CASE 1.—*Recurrent vertigo without history of otitis media, accompanied by tinnitus; attempt at section of the eighth nerve abandoned on account of poor exposure; destruction of the labyrinth in the temporal fossa without production of deafness; relief of vertigo but not of tinnitus, in spite of subsequent destruction of the cochlea.*

Alexander P., aged 44, was first seen by me at the Massachusetts General Hospital on Oct. 5, 1933. At that time he stated that he had been having constant tinnitus in the left ear, usually with headache, and attacks of extreme vertigo, vomiting and falling about three times weekly. He was completely incapacitated

for his occupation as an insurance salesman. The results of hearing tests were as follows:

Right		Left (affected ear)
20/25	whisper	12/25
20/30	conversation	4/35
23,000	upper limit	20,000
64	lower limit	96
25/12	Rinne	19/6
+	Weber	

The left drum was gray and slightly retracted. Neurologic examination, including lumbar puncture, gave negative results except for a positive reaction to the Romberg test.

Fig. 1.—Schematic view of the operation. The petrous ridge has been exposed by a subtemporal decompression. The superior canal has been opened with a round bur. Destruction of the ganglion can be completed by a brief coagulation.

As the Furstenberg diet had not been published at that time, section of the left eighth nerve was suggested to the patient and eagerly assented to. The operation was attempted on Oct. 11, 1933. The patient had a short, thick neck; his respiration was labored, and the eighth nerve was obscured by an overhanging vein. It seemed better to abandon the operation than to run the risk of hemorrhage or of injury to the brain stem from poor exposure. The wound was closed, and the patient was discharged.

The symptoms continued, of course, and the patient returned for treatment. As the posterior approach appeared closed, the subtemporal operation was devised. The situation was explained to the patient, and he assented to a trial of the new method. Accordingly, on April 23, 1934, with the patient under tribromethanol

anesthesia, an incision was made from the zygoma upward in the left temporal region. The squamous portion of the temporal bone was removed over an area about 4 cm. in diameter. The dura was elevated from the petrous portion, and by percussion the location of the superior canal was sought. One or two bur holes were made, through which only the bleeding mucosa of the mastoid cells was encountered; finally, however, the characteristic eburneous texture of the bony labyrinth was felt with the drill, and the superior canal was opened. A fine wire was introduced into its anterior end, and this wire was touched with the coagulating electrode, a light cutting current being used, for about five seconds. The dura was allowed to drop back into place, and the wound was closed.

The patient made an excellent recovery, after which he was free from attacks of dizziness, although at first he was somewhat unsteady on his feet. Hearing in the affected ear was substantially the same as before. Unfortunately, the tinnitus continued unabated.

An attempt was therefore made to destroy the remaining hearing in the hope of removing the tinnitus. The wound was reopened on May 5; the utricle was exposed by using a larger drill in the original hole, and coagulation was carried out for a longer period. Even this procedure failed to destroy all the hearing, and the tinnitus continued.

On May 8, therefore, the wound was opened once more, and the cochlea itself was exposed and lightly cauterized. This time the patient's hearing was destroyed, and temporary facial paralysis, which lasted for about six weeks, was brought on. The tinnitus was only modified, however, and it has persisted up to the time of writing. The patient has had no severe attacks of vertigo, although he occasionally feels slightly dizzy. The hearing in the right ear is deteriorating, as will be seen by the audiometer chart of Jan. 18, 1937 (chart 1). The residual symptoms have not been relieved by the Furstenberg diet, but the fact that the patient is receiving disability compensation may have something to do with this. He is at least extremely grateful for his operations.

CASE 2.—*Chronic recurrent attacks of vertigo and constant tinnitus for ten months following otitis media on the left side; subtemporal coagulation of the left labyrinth with partial relief of vertigo and later of the right labyrinth with almost complete relief; gradual loss of hearing in the right ear; persistence of tinnitus.*

Anna G., a single woman of 43, was first seen by me in consultation with Dr. Harry Solomon at the Boston Psychopathic Hospital in November 1934. About two years previously she had had an infection in the left ear, which had drained for two weeks and left the ear somewhat deaf. Ten months later she sustained a fall. Since then she had had a continuous dull headache. When the headache was at its worst she would have attacks of dizziness, with the sensation of objects spinning around her, lasting up to an hour. During some of these attacks she actually fell and often vomited. Six months before she came to the hospital, she began to have buzzing in the left ear. She became very depressed and entered the Psychopathic Hospital voluntarily. The diagnosis of a reactive depression was made there.

Neurologic examination gave negative results except for deafness of the left ear to both air and bone conduction. Both drums were found to be retracted. Caloric tests gave normal results in both ears. She was placed on a low salt diet and given ammonium chloride but was not relieved.

As the symptomatic attacks of dizziness and of tinnitus continued and were completely incapacitating, destruction of the left semicircular canals was undertaken on March 12, 1935, at the neurologic unit, Boston City Hospital. A small decompression was made in the temporal region. The dura was elevated to expose the mound marking the superior canal, and this canal was opened by a small bur

hole. A probe was inserted into the saccule, and a low intensity coagulating current was applied for about fifteen seconds. The dura was allowed to fall back into place, and the skin was sutured.

The patient made an excellent operative recovery. Hearing in the left ear was no worse than before according to tuning fork tests. Tinnitus and headache continued about as before, but the patient had no further attacks of vertigo for about a month. At the end of that time she had a severe attack, fell, and injured her scalp, so that it became infected.

She was therefore readmitted to the neurologic unit on Sept. 26, 1935. At this time audiometer tests were performed; they showed impairment of low tones on the left and, to a slight degree, on the right (chart 2). Caloric tests were carried out. Irrigation of the left ear with hot or with cold water produced slight nystagmus after about one and one-half minutes from both horizontal and vertical canals

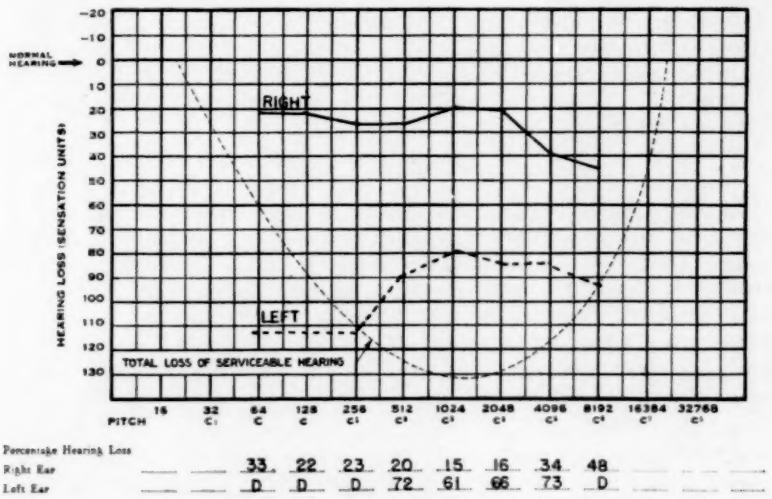

Fig. 2.—Audiogram for the patient in case 1 taken the last time he was seen, Jan. 18, 1937. Notice that hearing in the ear that was operated on (the left ear) was not entirely destroyed, despite attempts to coagulate the cochlear ganglion.

but did not produce dizziness at any time. Irrigation of the right ear produced nystagmus and dizziness after half a minute.

As the left labyrinth appeared incapable of originating vertigo on stimulation, and as there were mild deafness, tinnitus and retraction of the drum on the right, the only remaining step appeared to be to destroy the right labyrinth also. On October 26 the operation was carried out in exactly the same way on the right side as it had been on the left side.

The patient again made a good operative recovery. After operation there were temporary nystagmus and weakness of the right internal rectus muscle, both of which disappeared within two weeks. Audiograms indicated practically no change from the preoperative hearing condition. The patient was unsteady on her feet and had severe attacks of vertigo just before her discharge, on October 28. It was impossible to produce dizziness by irrigation of either ear. She became depressed and had to go to a convalescent home for some months.

Recently, however, there has been a marked improvement in her physical condition. After being confined to bed for two years, she is now able to be about,

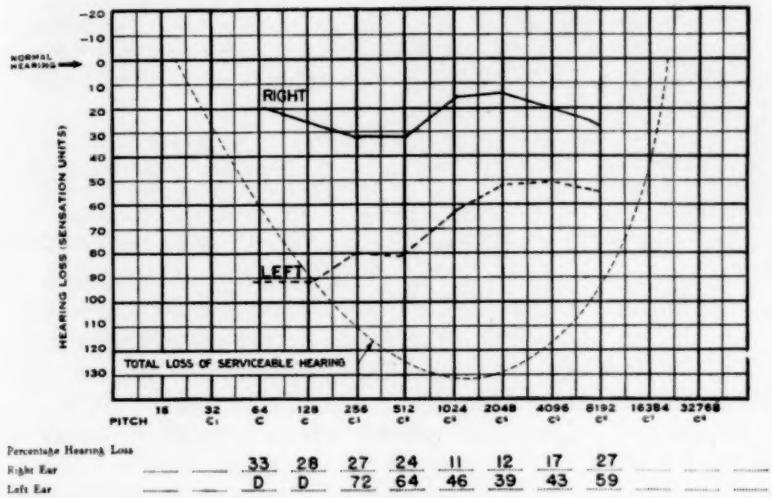
NORMAL HEARING
RIGHT
LEFT
HEARING LOSS (SENSATION UNITS)
TOTAL LOSS OF SERVICEABLE HEARING
PITCH
16 32 64 128 256 512 1024 2048 4096 8192 16384 32768
Percentage Hearing Loss
Right Ear 33 28 27 24 11 12 17 27
Left Ear D D 72 64 46 39 43 59

Fig. 3.—Audiogram for the patient in case 2 taken Sept. 26, 1935, after operation on the left ear but before operation on the right ear. Observe that hearing (which was impaired to an extent not accurately measured before the first operation) is retained in some degree in the left ear.

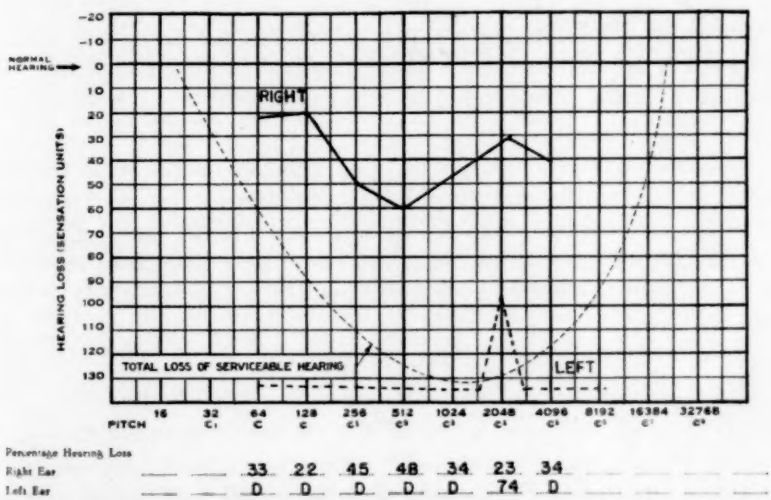
NORMAL HEARING
RIGHT
LEFT
HEARING LOSS (SENSATION UNITS)
TOTAL LOSS OF SERVICEABLE HEARING
PITCH
16 32 64 128 256 512 1024 2048 4096 8192 16384 32768
Percentage Hearing Loss
Right Ear 33 22 45 48 34 23 34
Left Ear D D D D D 74 D

Fig. 4.—Audiogram for the patient in case 2 taken at the time of her last report after operation on the right ear, Feb. 15, 1937. Notice that the deafness of the left ear has continued to progress spontaneously but that hearing in the right ear has suffered comparatively little from the operation.

though still slightly unsteady in walking. The tinnitus and the headache are little changed. She is mildly depressed, but grateful.

Audiograms (chart 3) made on Feb. 15, 1937, showed a general diminution of hearing in the right ear but by no means a complete loss. The left ear was almost totally deaf—evidently as a result of spontaneous progress of the original pathologic condition, for the loss was greater than at a previous examination.

COMMENT

The possibilities and also the limitations of the proposed operation are indicated by the 2 cases reported. In both, the function of the labyrinth and with it the severe attacks of vertigo have been done away with by a simple, safe operation, which need not affect hearing. The security of the cochlea is well illustrated by case 1, in which it was necessary to destroy almost the entire inner ear before hearing was finally reduced to negligible remnants. This very difficulty in attacking the cochlea, however, makes the operation ineffective against tinnitus. But the symptom of tinnitus often persists after section of the eighth nerve also.

Against what type of recurrent vertigo should such an operation be successful? It has already been pointed out that it ought to have the same scope as either section of the nerve or one of the older operations on the labyrinth itself. One of the cases reported furnishes an example of the so-called true Ménière's disease, or otosclerotic vertigo. In the other there was a history of otitis media on one side, but there were also evidences of a chronic degenerative process on the other side and the vertigo was not relieved until both sides had been operated on. The operation appears, therefore, to be effective against other types of aural vertigo as well.

Failures have been reported of other types of operation on the labyrinth. It is possible that they have been due to the poor exposure obtainable through the mastoid, for the function of the inner ear appears to be by no means easy to destroy. There may be reason to believe, therefore, that they can be avoided by use of the new technic in properly selected cases.

SUMMARY

An operation for the relief of recurrent vertigo is described. This operation consists in destruction of the vestibular ganglion through an opening into the superior canal from the upper anterior surface of the petrous ridge, approached by a small subtemporal decompression.

Five operations in 2 cases are reported; in each case the vertigo was relieved while hearing was preserved. In 1 of these there was no evidence that an infection of the ear had ever existed; in the other, there had been an infection on one side but both sides had to be operated on before relief was obtained.

TREATMENT OF LATERAL SINUS INFECTIONS WITHOUT OPERATION ON THE JUGULAR VEIN

E. MILES ATKINSON, M.D., F.R.C.S.
NEW YORK

To tie or not to tie: that is a question which has been exercising the minds of some otologists for several years. Since 1880, when Zaufal suggested ligature of the internal jugular vein in cases of thrombosis of the lateral sinus—a suggestion which on the face of it seemed logical enough—the orthodox teaching has been that if a thrombosed lateral sinus is found the internal jugular vein is to be ligated, and before the completion of the mastoid operation, lest the jarring of the work on the bone should displace portions of clot. Alexander [1] went even further and taught that if the diagnosis can be made with reasonable certainty before the operation, the first step should be ligature of the jugular vein. Many otologists, particularly those of the older school, still adhere rigidly to this teaching and ask why an operation which has given satisfactory results in the past should be tampered with. Whether ligature is essential or not, they say, they feel happier when the jugular vein is tied, and they prefer to continue in the old way. Well and good, but there are several considerations which give food for thought as to whether ligature is a necessity and suggest that this procedure may be another example of the *post hoc ergo propter hoc* fallacy.

THE ARGUMENT

In the first place, ligature of the internal jugular vein does not occlude all the paths whereby infection can spread.[2] There remain open collateral channels in the inferior petrosal sinus and the small veins which pass through the posterior condyloid foramina (venae condyloideae).

In the second place, the point of ligature may serve as another focus whence thrombosis can spread, and thus the feared extension may actually be fostered by the very means taken to prevent it. There are many

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Assistant surgeon, Manhattan Eye, Ear and Throat Hospital; formerly Hunterian Professor, Royal College of Surgeons of England.

This article was in the hands of the publisher before the Annual Session of the American Medical Association held in Atlantic City, N. J., June 7 to 11, 1937, at which the subject concerned was discussed.

1. Alexander, G.: Wien. med. Wchnschr. **73**:2293, 1923.

2. This paper is not concerned with the question of ligature versus excision of the vein, and jugular ligature is used as a general term to cover any operation for occlusion of the jugular vein.

records of spread of infection after ligature to previously uninfected areas, such as the deep fascial planes of the neck. Moreover, the thrombus which forms at the site of ligature may itself become infected, since it forms in a blood stream already containing organisms.

Thirdly, ligature of the internal jugular vein is not the easiest of operations under the conditions pertaining in such cases. With the lateral sinus occluded above, the vein is much smaller than normal, and it may be surrounded by a mass of inflamed glands, which add greatly to the difficulties. It is surprising with what skill the vein hides itself when it is being hunted, in contrast to the impish delight it takes in getting in the way when it is not. Ligature involves a definite addition to the length and the severity of the whole operation, a point of some moment in patients, particularly children, as seriously ill as those with a sinus infection. And a scar in the neck, a trifle if life is in the balance, is worth avoiding if it serves no essential purpose.

Such thoughts as these were approaching the saturation point when there occurred a case in my clinic which crystallized them out.

A. E., a girl of 14, was admitted to the hospital in a routine manner for an operation on the mastoid on account of aural discharge since scarlet fever at the age of 5 years. At operation extensive destruction of bone was found, and exposed in the cavity in the position of the lateral sinus lay a fibrous band. This band connected patent upper and lower portions of the sinus. The only explanation, apart from the unlikely one of a developmental abnormality, was that as a result of the infection the sinus had become infected, a thrombus had formed and organized, and spontaneous cure had taken place. No history of any severe illness or of rigors at any time could be obtained.

As a result of this case, I delved into the literature, to find not only that my ideas were not original but that some surprising statistics had been published on the subject.

THE LITERATURE

Körner, in the 1925 edition of his book, published some collected statistics [3] which showed that no matter whether the vein was ligated before or after opening the sinus or even was not ligated at all, the rate of recovery was virtually the same—around 60 per cent.

These figures suggest that it is immaterial whether ligature is performed or not, and that the result depends not so much on ligature as on the resistance of the patient.

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3. Körner (Die otitischen Erkrankungen des Hirns, Munich, J. F. Bergmann, 1925) gave the following collected statistics:

	Cases	D	R		Percentage
Sinus opened, no ligature:	132	55	77	=	58.3
Ligature before opening sinus:	92	36	56	=	60.8
Ligature after opening sinus:	68	29	38	=	56.7

In the same year Grünberg [4] published results which were even more startling. In his clinic the number of cases observed in the period from 1917 to 1923 was sixty-one. In twenty-one of these ligature was performed with a recovery rate of 15 per cent, while in forty it was not done, with a recovery rate of 60 per cent! These figures, taken at their face value, would suggest that ligature is a dangerous undertaking, but it is probable that the mortality rate in the first group is loaded by the inclusion of all the cases in which the condition was worst. Granting this, the second group still shows that a successful outcome is frequently possible without ligature, and it is interesting to see that the recovery rate is the same as that shown by Körner's collected statistics.

Stimulated by such statistics, Undritz [5] decided to make some experimental observations. He produced thrombosis of the lateral sinus in twenty-two dogs. In eleven he tied the jugular vein, and in eleven he left it untied. The results were exactly the same—in each group five died and six recovered, a recovery rate of 55 per cent, again very close.

The conclusions drawn by Undritz were (1) that ligature of the vein exercises no noticeable influence on the course of the disease, (2) that experimental sinus thrombosis shows a tendency to spontaneous recovery and (3) that the thrombus is to be regarded as a protective mechanism not lightly to be interfered with. All his healed sinuses showed the same appearance—the lumen of the sinus obliterated by fibrous tissue closely incorporated with the wall of the vein, precisely the state of affairs in the case described.

THE PROCEDURE

In view of these discoveries, I decided that I would throw in my lot with the heretics and see what came of abandoning the idea of ligature of the jugular vein as a sine qua non in cases of sinus thrombosis. The method adopted was to expose the sinus to the limits of the pathologic process in the wall and, in the absence of evidence of an occluding thrombus or of a general infection, to do no more—so far quite orthodox. If, however, a thrombus was present, the sinus wall was necrotic or there were indications before operation of a general infection, then the sinus was opened and obliterated but without ligature of the jugular vein. Only if signs of general infection continued or if local signs of involvement of the jugular vein appeared was the operation on the neck undertaken. It will be seen that it was performed twice only. It is important at this point to make clear my mental attitude,

4. Grünberg, K.: Monatschr. f. Ohrenh. **59**:3, 1925.
5. Undritz, W.: Ztschr. f. Hals-, Nasen- u. Ohrenh. **13**:204, 1926.

which was purely tentative and experimental. No unnecessary risk was to be taken. In case of doubt I inclined toward ligature, the devil I knew, rather than away from it. Those five cases, therefore, in which ligature was not employed can be understood to have caused no undue anxiety.

THE MATERIAL

The number of cases, fifteen only, is too small to have any statistical value, and no attempt will be made to draw such conclusions from them. For instance, by some freak of chance, all the patients recovered—and a mortality rate of nil with this complication is obviously absurd. The material has certain points of merit, however, in that it comprises every case of involvement of the lateral sinus, not necessarily thrombosis, which was observed in my clinic in England (eleven) or in my private practice (four) in the three year period from the middle of 1932 to the middle of 1935. There has been no selection. Every patient was operated on, and the after-treatment ordered, supervised and in private cases carried out, by myself, so that there was an absolute uniformity of method. The mistakes were mine as much as the successes. I believe that a small series carefully studied by one person can teach lessons as valuable as those to be learned from a larger series less uniformly treated, and it is in this belief that I offer this paper. At least I have clarified my own ideas in the writing of it, as I have gained invaluable experience in collecting the material and have been confirmed in my heterodoxy by the results.

The cases arrange themselves into three groups:

1. Cases in which there were granulations on the sinus wall without demonstrable thrombosis—8.
2. Cases in which there were inflammatory changes in the sinus wall with gross thrombus formation—5.
3. Cases in which there was necrosis of the sinus wall without thrombosis—2.

Group 1.—Cases in which there were granulations on the sinus wall without demonstrable thrombosis.

Of the eight cases in this group, in five the sinus was found lying pathologically exposed in the mastoid cavity at the time of operation, while in three it was still covered by the bony plate which concealed a perisinus abscess. The common factor was that in every case the sinus was in contact with pus, and inflammatory changes had occurred in the wall of the vessel. The treatment was to expose the full extent of the disease in the wall of the sinus and to avoid any interference with protective granulations. In the absence before operation of

rigors or a positive blood culture, the sinus was not opened. The syringe as a test of patency was never employed, because the information so obtained not only is valueless but may be misleading, aspiration of blood being possible in the presence of a thrombus. Further, the point of aspiration can serve as another possible *point d'appui* for the infection. The only departure from orthodoxy, if such it may be called, was that in borderline cases the sinus was given the benefit of the doubt and not opened. On one occasion only was this faith not justified, so that a further procedure had to be undertaken (case 14).[6]

These results give cause for thought on the question of mural thrombosis. In some of these sinuses there must have existed a mural thrombus at the site of infection, though in how many it is, of course, impossible even to guess. This condition has been made into a bugbear, and its danger would seem to have been greatly exaggerated. Thrombosis is a protective mechanism, and if a mural clot is left undisturbed it will continue to exercise its protective function with great success.

Group 2.—Cases in which there were inflammatory changes in the sinus wall with gross thrombus formation.

This group comprises cases in which the condition is of varying degrees of severity, depending partly on the virulence of the organism and partly on the length of time during which the sinus has been subjected to infection. There are three stages of the process.

1. The wall of the sinus is thickened and covered with lymph. The sinus feels solid and on being opened is found to contain a white gelatinous clot, which is firmly attached to the wall of the vessel but which can be removed readily and in one piece. This is the stage in which the condition can undergo spontaneous cure and in which the prognosis should be good. In the one case of this stage in this series there was a perfectly straightforward course after operation despite the fact that the jugular vein contained clot down to the clavicle (case 10).

6. Since this paper was written, there has come my way one more case which belongs in this group and in which recovery was uneventful (clinic of Dr. Marvin F. Jones at the Manhattan Eye, Ear and Throat Hospital). A man with a history of eight weeks' discharge from the ear and continuous and increasing throbbing pain around it for three days was found to have a large perisinus abscess, with granulations extending into a superjacent cell, where they were actually necrotic. The exposed sinus showed no evidence of thrombosis. An attempt at the usual full exposure was abandoned because of the thickness of the skull, as it was felt that the benefit likely to be derived was not commensurate with the damage which might be done by the inevitable jarring from work on such thick bone. The wound was packed open, but as nothing untoward happened secondary suture was done eight days later, and the patient left the hospital within two weeks with the wound healed and the middle ear dry.

2. The sinus wall is soft and edematous, dark red and flaked with lymph. Within is a red friable thrombus loosely attached to the wall of the sinus and liable to break into pieces during removal.

3. This stage is really only a late phase of stage 2. The wall of the blood vessel has become necrotic and the clot is beginning to break down, so that frank pus may be found actually in the lumen of the sinus (cases 9 and 11). It is in the last two stages that the process is liable to spread to the jugular bulb and to be the cause of pyemic signs (cases 12 and 13).

The treatment in all three stages is essentially the same. The clot is removed completely from the upper end, but from the lower end only so much is removed as can be pulled out by gentle traction with forceps. If this results in the whole clot coming away, with free bleeding, so much the better. If not, no attempt is made to clear the bulb for fear of breaking up an organizing thrombus. No operation is done on the vein unless it is thought to contain clot. The prognosis in the last two stages is always anxious, and it is in these stages, especially when thrombosis has spread to the bulb, that ligature of the jugular vein may prove necessary. In case 13 the vein was tied after the third postoperative rigor, and there were no more. It is difficult to believe that this result is anything but *propter hoc,* and it is for such cases and such cases only that I believe ligature of the jugular vein will be reserved in the future.

Group 3.—Cases in which there is necrosis of the sinus wall without thrombosis.

In two cases the condition was of this dangerous type. The infection passes to the sinus plate and thence to the sinus wall, which becomes adherent to it without the formation of an intervening abscess. An infection of the blood stream of septicemic type is thus produced, and it is probable that in many cases so-called aural bacteremia (Lake and Peters [7]) has its origin in this way.

The reason for the great danger lies in the difficulty of removing the source of infection. As the infected bone is removed, the adherent wall of the sinus comes away with it, with consequent severe hemorrhage, and it is possible for a great deal of blood to be lost in the attempt to get beyond the infection. Moreover, ligature of the jugular vein is obviously useless under such circumstances, so that one is forced to rely on as complete a clearance of infected bone as is possible and safe and on obliteration of the sinus. The prognosis is bound to be grave, and for both patients to recover is more than one has any right to expect.

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7. Lake, R., and Peters, E. A.: Handbook of Diseases of the Ear, ed. 5, New York, William Wood & Company, 1927.

THE INDICATIONS FOR LIGATURE

The signs on which I have based a decision to operate on the vein are three:

1. Tenderness along the course of the vein is constantly present if thrombosis has extended into the vein, but I have yet to feel the classic "cord in the neck." Palpation should be of the gentlest lest a portion of clot be dislodged; indeed the patient usually insists that it shall be.

2. The presence of palpably enlarged glands, though not an invariable nor an infallible sign of phlebitis and thrombosis, is a significant one. It prompts exploration unless there is some other good reason for the lymphadenitis.

3. Repeated rigors after operation on the sinus suggest a friable clot in the bulb. As a general working rule, a patient is allowed two rigors after operation in case a portion of thrombus has been loosened by the manipulations. A third leads to exploration of the jugular vein, removal of any clot found, excision of a portion including the common facial vein, and suture of the upper end to the skin to provide drainage of the bulb. Exposure of the bulb itself in the jugular foramen is a severe and dangerous operation, which can seldom be necessary. I have never felt called on to attempt it.

THE RESULTS

The total number of cases is fifteen.

Thrombosis of the sinus was present in five (cases 9 to 13).

Operation on the jugular vein was necessary in only two (cases 9 and 13)—in case 9 probably only because the condition had been allowed to progress too far before operation, so that thrombosis had already extended into the vein, and in case 13 because of repeated rigors from a disintegrating clot in the jugular bulb.

Necrosis of the sinus wall without thrombosis but with infection of the blood stream and positive blood culture was found in two cases (cases 14 and 15).

Perisinus abscess or pathologic exposure of the sinus was found in nine cases, in which treatment was by full exposure. Further operation on the sinus was necessary in one (case 14).

Mortality was nil, although one patient (case 12) had the additional complication of a cerebellar abscess.

THE CONCLUSIONS

1. Ligature of the jugular vein need not be an invariable accompaniment of an operation on the lateral sinus.

2. Ligature of the jugular vein has its most valuable application when thrombosis has extended to the jugular bulb.

3. Infection of the lateral sinus has a marked tendency toward spontaneous cure if infected tissue is removed and free drainage provided.

THE CASES

Brief notes follow of the fifteen cases on which this paper is based:

Group 1.—Perisinus abscess or pathologic exposure of the sinus.

CASES 1 to 8.[8]—These are not worth individual reports, and the method of treatment and the results have been described.

Group 2.—Thrombosis of sinus.

CASE 9.—R. M., a girl aged 12 years, had an acute process of three weeks' duration. At operation the sinus was found exposed and the wall necrotic, the lumen containing a red thrombus and pus. The sinus was cleared of clot and obliterated; no operation on the jugular vein was done. Recovery followed. Bacteriologic examination showed Streptococcus haemolyticus.

CASE 10.—E. S., a girl aged 11 years, presented an interesting condition. She was admitted for symptoms of complication involving the chest after a cold; the right ear was running; there was no tenderness over the mastoid; the temperature was 103 F., the respiratory rate 38 and the pulse rate 124. Mastoidectomy was performed one week later because of insufficiency of signs of pulmonary involvement and revealed sinus thrombosis. The sinus was cleared of clot. Continued signs of involvement in the chest and high temperature were present, but no rigors. Five days later, on account of two attacks of cyanosis and commencing pain in the right side of the neck, a second operation was performed and the jugular vein was found thrombosed to a point behind the clavicle; the clot was removed. Uninterrupted recovery followed. Bacteriologic examination showed Streptococcus pyogenes.

CASE 11.—B. G., a woman aged 59, had acute exacerbation of a chronic process, with signs of perilabyrinthitis but no rigors. Operation revealed a perisinus abscess, a necrotic sinus wall and a red thrombus beginning to disintegrate. The upper end was cleared, with free bleeding; the lower end showed no bleeding. The jugular vein was not ligated. The postoperative course was uneventful, with no rigor. Bacteriologic examination showed Str. haemolyticus.

CASE 12.—R. R., a boy aged 10 years, had acute exacerbation of a chronic process and was sent to the hospital three weeks later with signs of cerebellar abscess. At operation the sinus was found exposed and thrombosed, and a firm clot was removed, with no bleeding from the lower end. The cerebellar abscess was drained. The jugular vein was not ligated. Recovery followed. Bacteriologic examination showed Str. pyogenes and Bacillus coli communis.

CASE 13.—J. W., a man aged 35, had acute exacerbation of a chronic process. The first rigor occurred on his admission to the hospital. No tenderness or lymphadenitis in the neck was noted. Operation was performed immediately and revealed widespread disease, softening of the sinus plate, a perisinus abscess and a thrombosed sinus. A firm red clot was removed, with free bleeding from the

8. Cases from the Department of Otolaryngology, Royal United Hospital, Bath, England, and from my private practice.

upper end but none from the lower. No postoperative rigor occurred until the fourth day after a dressing; a rigor occurred again on the sixth day and on the eighth. Operation for ligature of the jugular vein revealed a breaking-down clot in the bulb, none in the vein. The upper end was sewed to the skin and washed out daily. No further rigor occurred. Recovery followed. Bacteriologic examination showed Str. pyogenes and Staphylococcus aureus.

Group 3.—Necrosis of wall without thrombosis.

CASE 14.—A. J. D., a woman aged 53, had an acute process of four weeks' duration. At operation the sinus was found exposed, with granulations on the wall; removal of the adjacent bone showed a normal wall. No further intervention on the sinus was attempted; the wound was left open. Three days later signs of perilabyrinthitis developed with mental confusion and wandering; then she had a rigor on three successive days. There was no leukocytosis; a blood culture remained sterile. Queckenstedt's sign could not be elicited, and the spinal fluid was normal, except for increased pressure. At operation necrosis of the sinus wall was found; attempted removal of bone caused profuse hemorrhage from tearing of the wall; no thrombosis was present; the sinus was exposed as widely as possible and packed off. The jugular vein was not ligated. No further rigor occurred. A third operation, decompression for localized nonsuppurative encephalitis, was ultimately performed. Recovery followed. Bacteriologic examination showed Str. haemolyticus.

CASE 15.—M. P., a girl aged 14, had an acute process of five weeks' duration. When the mastoid was exenterated, the sinus plate was normal and the sinus was not exposed. The postoperative course was unsatisfactory; three weeks later a rigor occurred on two occasions. Blood culture showed Str. haemolyticus. At operation soft bone was found over the sinus; the adherent sinus wall came away with the bone; no further rigor occurred. Recovery followed.

570 Park Avenue.

OTITIC SEPSIS

ANALYSIS OF TWENTY-ONE CASES AND ANATOMIC STUDY

ARTHUR L. JUERS, M.D.
OWENSBORO, KY.

The problem of otitic sepsis is one which frequently taxes the diagnostic acumen of the otologist to the utmost and necessitates the use of extensive therapeutic measures if optimum results are to be obtained. The literature on this subject is at times confusing, particularly with reference to the nature of the pathologic process in the sigmoid sinus. The terms sinus thrombosis and sinus phlebitis are unfortunately at times used interchangeably. The broader term "otitic sepsis" indicates an infection of the blood stream secondary to infection in the middle ear or the mastoid process. At the point of entrance of infection into the blood stream—usually the sigmoid sinus—there is always phlebitis, and thrombus may or may not be present.

The opinion of otologists as to the proper management of otitic sepsis is fairly uniform as far as the mastoid process and the sigmoid sinus are concerned. However, there is still some divergence of opinion as to whether and when ligation of the internal jugular vein should be done. The purpose of this study is to bring out any possible additional evidence which will help to answer this question and to present an anatomic study of the sigmoid sinus, which is of interest in connection with this subject. There is nothing new to be said as to symptoms, diagnosis or laboratory studies. However, these phases of the subject will be briefly touched on as concerns the group of cases of otitic sepsis to be reported on.

SYMPTOMS AND DIAGNOSIS

The outstanding clinical feature of sepsis complicating otitis media and mastoiditis is the septic, "picket-fence" type of temperature curve. When the temperature maintains a sustained high level it indicates the development of meningitis or overwhelming sepsis with metastatic lesions. A chill may or may not precede the elevation in temperature, this manifestation apparently depending on the age of the patient (table 1). The 7 patients over 15 years of age all had a definite chill

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From the Otolaryngology Service, University of Minnesota.

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before each rise in temperature. There were 9 patients, aged from 10 to 15 years. Of these, 3 had chills and 6 had no chills. Those below 10 years of age had no chills. None of this last group had at the onset of the sepsis the convulsions occasionally seen in children. There

TABLE 1

Case No.	Age, Yr.	Chill	Culture of Blood	Pathologic Condition of Sinus	Surgical Treatment	Comment
1	1	No	Not taken	Phlebitis	Exposure of sinus	
2	1½	No	Not taken	Phlebitis	Exposure of sinus	
3	4	No	Not taken	Phlebitis	Exposure of sinus	
4	5	No	No growth	Mural thrombus	Obliteration of sinus; jugular ligation	
5	8	No	Hemolytic streptococcus	Mural thrombus	Obliteration of sinus; jugular ligation	
6	10	No	Hemolytic streptococcus	Occluded thrombus	Thrombectomy; jugular ligation	
7	10	No	No growth	Phlebitis	Exposure of sinus	
8	10	Yes	Hemolytic streptococcus	Phlebitis	Exposure of sinus	
9	12	No	Contaminated	Occluded thrombus	Thrombectomy; jugular ligation	
10	12	Yes	Hemolytic streptococcus	Occluded thrombus	Thrombectomy; jugular ligation	Patient died of meningitis; thrombosis beyond torcula
11	13	No	Hemolytic streptococcus	Occluded thrombus	Mastoidectomy only	Sinus thrombosis and localized meningitis at autopsy
12	14	Yes	Hemolytic streptococcus	Occluded thrombus	Incision of sinus; jugular ligation	Patient died from prolonged suppuration of joint
13	15	No	No growth	Phlebitis	Exposure of sinus	
14	15	No	Pneumococcus	Phlebitis	Exposure of sinus; jugular ligation	Endocarditis and multiple abscesses at autopsy
15	16	Yes	Staphylococcus; hemolytic streptococcus	Mural thrombus	Obliteration of sinus; jugular ligation	
16	16	Yes	Hemolytic streptococcus	Occluded thrombus	Thrombectomy	
17	17	Yes	Hemolytic streptococcus	Mural thrombus	Obliteration of sinus	
18	18	Yes	Hemolytic streptococcus	Mural thrombus	Obliteration of sinus; jugular ligation	
19	20	Yes	No growth	Phlebitis	Exposure of sinus	
20	21	Yes	No growth	Phlebitis	Exposure of sinus; jugular ligation	Pleural effusion, cerebellar abscesses and meningitis at autopsy
21	40	Yes	No growth	Broken down thrombus	Drainage of sinus through mastoid cavity	

was usually marked irritability. One child, aged 8 years, seemed to be of perfectly normal general appearance, in spite of a daily elevation in temperature to 104 F. and the presence of hemolytic streptococci in a culture of the blood. There was no correlation between the presence of chills and the presence or absence of organisms in cultures of the

blood. In 4 cases in which culture of blood gave positive results there were no chills, and in 3 in which they were negative there were chills. Two patients with occluding thrombi had no chills.

The mental changes were varied and ranged from no symptoms at all to drowsiness, irritability or mild delirium. Headache was frequently present. The local physical findings were dependent on the nature of the pathologic picture of the mastoid and the location of the sinus involvement.

In only 4 cases did the factor of positive results of culture of the blood per se alter the therapy. In all other cases the temperature curve and the clinical appearance of the patient warranted surgical intervention before positive results were obtained. With 3 children no culture of blood was made, because it was felt that the possible information to be gained would not justify the disturbance created from the standpoint of the child. This point has been stressed by Lillie.[1]

The Ayer-Tobey [2] modification of the Queckenstedt test was of value in determining which sinus was involved in a case of bilateral mastoiditis. In several cases it gave useful information as to the patency of the sinus. In 1 case it was definitely misleading.

Roentgen examination of the mastoid process is of value in determining what type of mastoid structure is being dealt with and obtaining some information as to the nature of the pathologic process existent in the mastoid. In 1 case of early bilateral involvement the roentgenogram showed early decalcification in the posterior angle of 1 mastoid. At operation it was verified that this was the point at which infection was entering the blood stream.

The leukocyte and the differential count usually give corroborative information of severe infection, although there are occasional exceptions, which will be noted later. The temperature curve gives a more accurate and reliable indication of the presence and the severity of the sepsis.

STUDIES OF THE BLOOD

Culture of the blood gave positive results in 11 of the 21 cases (table 1). In 3 cases no cultures were made, for reasons already stated. In case 15 the first culture showed the presence of staphylococci. The temperature gradually subsided, but two weeks later the patient had another chill and septic episode. A culture of blood at that time revealed hemolytic streptococci. The original mastoid culture showed both staphylococci and streptococci. The results of culture of the blood in

1. Lillie, H. I.: Suppuration of the Temporal Bone Accompanied by Infection in the Blood Stream, Arch. Otolaryng. **18**:630 (Nov.) 1933.

2. Ayer, J. B., and Tobey, G. L., Jr.: Dynamic Studies on the Cerebrospinal Fluid in the Differential Diagnosis of Lateral Sinus Thrombosis, Arch. Otolaryng. **2**:50 (July) 1925.

this case would indicate either that two different types of organisms entered the blood at different times or that one of the cultures gave positive results owing to outside contamination.

In case 15 there was a white blood cell count of 7,100, with 71 per cent neutrophils, at the time of admission. Four days after thrombectomy and ligation of the internal jugular vein, when the temperature was still septic, the leukocyte count was 4,200, with 76 per cent neutrophils. In case 21 the patient had had chills one week prior to admission, and the leukocyte count at the time of admission was 5,950, with 62 per cent neutrophils. The next day the count was 8,850, with 91 per cent neutrophils. Unfortunately, Schilling's differential counts were not done as a routine with these patients. However, it is of interest to note that on routine blood counts leukocytosis was absent in three of the four counts made for these 2 patients, both of whom had sinus thrombosis with sepsis. Repeated daily counts might have shown a higher degree of leukocytosis in these patients, but the clinical course of the patient must be given preference to routine laboratory studies in evaluating the progress of the sinus disease. In all the other cases there was the usual leukocytosis.

TREATMENT

A. *Mastoid and Sigmoid Sinus.*—Complete exenteration of all accessible mastoid cell structure is a prerequisite to good results in all mastoid surgical procedures. Cell structure is most frequently overlooked in the posterior angle, medial to the digastric crest, and in the zygomatic region. In the presence of otitic sepsis the wall of the sinus should be uncovered in all directions, from the point of entrance of the infection into the sinus, if possible, until normal-looking wall or dura is encountered. If necessary, the entire sinus should be exposed to locate the seat of the phlebitis or thrombosis. If the Ayer-Tobey test has indicated that the sinus is thrombosed or the appearance of the wall of the sinus suggests it, incision after adequate exposure of the sinus is indicated at the time of the mastoidectomy. If the pathologic process appears to be just phlebitis, wide exposure of the involved area may suffice and the patient can be observed further postoperatively. If progress is not satisfactory, a secondary exploratory operation should be done after a preliminary Ayer-Tobey test. Aspiration of the sinus to determine if thrombosis is present can be relied on only if no blood is obtained. If in a septic patient a thrombus is present, it should be completely removed and free bleeding obtained from above and below. A suction cannula is the safest means with which to remove the extreme upper and lower ends of the thrombus. The diseased wall of the sinus may be excised.

The proper time for removing the packing depends on the clinical course of the patient. If the sepsis has rapidly cleared, the packing may be removed after about seven or eight days. By this time the open ends

of the sinus should be firmly thrombosed. If sepsis has continued postoperatively, the packing should be removed in three or four days and the open ends of the sinus further explored with suction. This revision of the mastoid cavity and sinus was done twice in cases 6, 15 and 18. In 2 instances pus was evacuated from the open end of the sinus. Definite clinical improvement followed this procedure each time. Maximum drainage may be established by packing the wound wide open. A secondary closure may be made later if necessary. It is sometimes advantageous to pack off a partially thrombosed sinus and thereby produce complete obstruction at a secondary operation. This was done in case 17 and there was immediate cessation of the septic temperature. Ligation of the internal jugular vein was not done in this case.

B. *Internal Jugular Vein.*—Rott,[3] whose views on treatment of infection of the lateral sinus are the same as those of Dixon and others, has drawn the following conclusions:

1. The only indication to be met in the treatment for lateral sinus infection is the prompt removal of the infected area, whether that area is in the mastoid cells, the sinus plate, the wall of the sinus or inside the lumen of the sinus.

2. Ligation of the jugular vein does not prevent absorption of toxins and bacteria into the general circulation and hence is a superfluous procedure.

3. Ligation of the jugular vein should be reserved for definite infection in the vein, and then should be accompanied by resection.

Coates reviewed 14 cases from his service and concluded, "in case of doubt ligation is advisable—then everything possible surgically has been done."

In Meltzer's [4] series of cases metastasis appeared four times as often after ligation as before. The author made cultures of blood in 65 cases; in 50 of these results were negative before ligation. Of these 50 results became positive in 13 after ligation. It is difficult to draw any conclusions from these statistics, because ligation is frequently done at a time when the patient is not improving clinically. Obviously, culture of the blood is more likely to give positive results at such a time than earlier in the course of the disease. However, it would seem that ligating the jugular vein does not promptly end continued sepsis.

Lillie [1] reviewed 52 cases of otitic sepsis. For phlebitis of the sigmoid sinus in hemorrhagic mastoiditis his procedure consisted of adequate exposure of the area of phlebitis. Of 11 cases in which primary obliteration of the sigmoid sinus was carried out because of thrombosis, the jugular vein was subsequently ligated in 4. The author stated the

3. Rott, O. M.: Why Ligate Jugular Vein in Cases of Lateral Sinus Thrombosis? Arch. Otolaryng. **14**:272 (Sept.) 1931.

4. Meltzer, P. E.: Treatment of Lateral Sinus Thrombosis, Arch. Otolaryng. **22**:131 (Aug.) 1935.

opinion that transfusion seems to have a more beneficial effect than ligation of the jugular vein, but that as ligation is an accepted form of treatment, its judicial use may avoid medicolegal complications. Primary obliteration of the sigmoid sinus and primary ligation of the jugular vein were performed in 5 cases in which symptoms of sepsis were sufficiently severe to warrant primary operation on both the sigmoid sinus and the jugular vein. Under the heading "Secondary Operative Intervention for Infection of the Blood Stream" the author reported 2 cases in which obliteration was done first and ligation or resection later and 14 cases in which obliteration and ligation were done simultaneously.

Maxwell [5] stated,

> In several of our cases in which the vein was undisturbed at the original operation sepsis persisted for from three to six days only to subside promptly after the section of the internal jugular vein. This operation should not be abandoned, because there is no doubt as to its value in the majority of cases.

He did not say whether at the time of the ligation at a secondary operation any revision of the mastoid cavity or the sigmoid sinus was done. This point will be discussed later.

RESULTS OBTAINED IN THIS SERIES OF CASES

For this discussion cases will be classified according to the pathologic picture in the sinus, namely, as instances of phlebitis, mural thrombus and occluding thrombus.

A. *Phlebitis.*—There were 9 cases of phlebitis. Seven of the patients recovered after complete mastoidectomy and adequate exposure of the sigmoid sinus. The patient in case 14 died forty-eight hours after admission. Mastoidectomy and wide exposure of the sinus were done shortly after admission. There was no improvement, and the next day ligation was done. Necropsy revealed endocarditis, multiple visceral abscesses and evidence of severe sepsis. No change in surgical treatment would have produced a favorable result. In case 20 mastoidectomy and exposure of the sinus were performed the day after admission. There was no improvement, and ligation was done two days later. Aspiration of the sinus showed the presence of blood. The patient became gradually worse and died a few days later. A small cerebellar abscess, meningitis, multiple pleural abscesses and bilateral pleural effusion were found at autopsy. Early obliteration of the sinus might have been of value in this case. The mortality in cases of phlebitis was 22 per cent.

B. *Mural Thrombus.*—Under this heading were 5 cases. The condition in case 4 promptly cleared after primary obliteration of the sinus

5. Maxwell, J. H.: Thrombosis of the Sigmoid Sinus, Arch. Otolaryng. **25**: 184 (Feb.) 1937.

and ligation of the jugular vein. In case 5 septic temperature continued for several days after obliteration of the sinus and ligation. In case 15 there were the unusual bacteriologic findings already mentioned. Obliteration and ligation in this case did not end the sepsis, and revision of the mastoid cavity and the sinus was done twice. Sepsis in case 17 ceased after obliteration of the sinus at a secondary operation. Revision of the sinus was necessary in case 18 after secondary obliteration and ligation.

C. *Occluding Thrombus.*—Seven cases came under this heading. In 3 of these at the time of thrombectomy and ligation the vein was found collapsed. In 1 of these 3, case 6, sepsis continued, and reexploration of the sinus revealed pus in the upper end. Improvement followed evacuation of this. Pleurisy developed in case 9 after thrombectomy and ligation. In case 12 there was a metastatic lesion in one hip at the time of admission, and death occurred several weeks later as a result of continued suppuration in this region. Of the remaining 4 cases, ligation was done in 1, case 10. The patient was just recovering from pneumonia of three weeks' duration and died of meningitis several days after operation. In case 11 there was septic temperature for two days after a simple mastoidectomy, and then conditions suggestive of abscess in the temporal lobe developed. Consultation with the neurologic service resulted in a decision to wait until the suspected abscess was walled off before performing any further operation. The patient died a few days later, and autopsy showed sinus thrombosis and localized meningitis over the temporal lobe. Had the septic temperature early been given due consideration, adequate surgical intervention might have produced a favorable result. In case 21 there was a broken-down thrombus, and recovery followed drainage of the sinus through the mastoid cavity. In case 16 there was thrombosis extending from the knee of the sinus to the jugular bulb. Thrombectomy was done, and free bleeding was obtained from above but not below. The temperature remained normal after the first postoperative day. This excellent result without ligation makes it questionable whether ligation is always necessary when free bleeding is not obtained from the bulbar end of the sinus.

The mortality in the cases of thrombosis was 25 per cent.

Small transfusions at intervals of a few days were given in all cases during the period of sepsis.

MORTALITY

The general mortality in this series of cases was 24 per cent. Lillie [1] reported a mortality of 16.3 per cent in his series. Others have reported figures ranging from 25 to 35 per cent.

Apparently little is being done with specific serum therapy for septicemia in an effort to lower the mortality rate. Convalescent serum is

sometimes used, and occasionally donors are given specific immunization. The results with these procedures have, apparently, not been startling. Scott,[6] in a review of 311 cases of septicemia due to various causes, reported a general mortality of 74 per cent. Only cases in which a culture of the blood showed staphylococci and streptococci were included. Cadham[7] reviewed 100 similar unselected cases in which the results of culture of the blood were positive and in which specific serum therapy was used and reported a mortality of 15 per cent. Among cases in which the condition was of otitic origin there was a mortality of 10 per cent. His method of serum therapy is different from that generally employed:

> A large number of rabbits are immunized against a specific strain of streptococcus or staphylococcus. The organism obtained from the patient is identified as to strain and a vaccine is produced and injected into several rabbits which have been immunized to the same strain. The next day 10 cc. of blood is removed from the rabbit, and the serum from this is injected into the patient.

Instead of a transfusion of whole blood Cadham advocated the use of serum only from a compatible donor, as the cellular content of whole blood, according to the author, lowers the amount of complement of the patient's blood. In view of the extremely low mortality obtained in the cases in which this method was employed it seems that it should be given more extensive trial to determine if similar results can be obtained by other workers.

COMMENT

Few otologists now do ligation as a routine procedure in all cases. The present tendency is first to carry out the necessary operation on the mastoid and the sinus and then to observe the case further. If progress is not satisfactory, ligation is usually considered in order. If ligation is done as a secondary procedure, the packing of the mastoid cavity and that of the sigmoid sinus are usually revised at the same time. After this the patient is usually given a blood transfusion. If the patient then makes rapid clinical improvement, there are three procedures which may be given credit for the favorable result, namely, ligation, revision of the mastoid cavity and sigmoid sinus and transfusion. These must be taken into consideration when evaluating the benefit derived from doing ligation as a secondary procedure.

Ligation of the jugular vein was done in 10 cases of this series. Eliminating the 4 of these cases in which the condition terminated fatally, I shall discuss only 6. Of these 6, the vein was found collapsed

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6. Scott, W. J. M.: Principles of Treatment of Septicemia, J. A. M. A. **105**: 1246 (Oct. 19) 1935.

7. Cadham, F. T.: Septicemia: Method of Treatment, Am. J. M. Sc. **188**: 542, 1934.

in 2. It is certainly doubtful whether ligation of a collapsed vein is of any value. While in the remaining 4 cases the usefulness of ligation cannot be disproved, the clinical course did not indicate that any appreciable benefit resulted from ligation. In view of the good results obtained in a few other cases with a similar pathologic picture when no ligation was done, it is my impression that ligation of the internal jugular vein is a procedure of little value in the management of otitic sepsis unless there is thrombosis in the vein itself.

Ottenberg [8] made cultures of blood from both internal jugular veins in a case of sinus thrombosis and found that on culture there were more colonies of bacteria per cubic centimeter in the blood obtained from the normal side. Dixon [9] has pointed out that in cases of sinus thrombosis the infection enters the blood stream chiefly from the torcular end of the thrombus and from the emissary connections of the sinus.

ANATOMIC CONSIDERATIONS OF THE SIGMOID SINUS

The emissary connections of the sigmoid sinus and the jugular bulb are stressed particularly by those otologists who feel that ligation of the internal jugular vein is an unnecessary procedure in the surgical treatment of phlebitis or thrombosis of the lateral sinus. In reviewing the literature on this subject, I found that no attempt had ever been made to correlate the position of the sigmoid sinus and the extent of its collateral circulation. It occurred to me that if the anterior or the posterior position of the sinus gave an indication of the amount of its collateral circulation, then its position as found at operation might give the surgeon some idea as to whether the value of ligation of the jugular vein would be offset by an abundant collateral circulation of the sinus or not.

The superior and the inferior petrosal sinus are constant anatomic structures, and under certain conditions can serve as channels for the exit of blood from the sigmoid sinus and the jugular bulb. The emissary veins passing through the mastoid and the posterior condyloid foramen, on the other hand, are somewhat variable in size and not infrequently absent. The only measurements easily available are those of the foramina through which these veins pass. The size of the foramen does not always supply an accurate idea as to the size of the vein, as a fair-sized emissary foramen may be largely filled with loose connective tissue and have a small vein or none present. An estimate of the size of the foramina, however, would show the maximum amount of collateral circulation possible if the veins filled them entirely. This fact

8. Ottenberg, in Braun, A.: Sinus Thrombophlebitis, New York, Paul B. Hoeber, Inc., 1928.

9. Dixon, O. J.: Non-Ligation of Internal Jugular Vein in Sigmoid Sinus Thrombophlebitis, Tr. Am. Laryng., Rhin. & Otol. Soc. **38**:142, 1932.

must be taken into consideration in an estimate of the probable collateral circulation of the sigmoid sinus in terms of the size of the previously mentioned foramina.

The best statistics available on measurements of the mastoid and the posterior condyloid foramen are those of Streit [10] and of Denker and Kahler.[11] Their measurements closely paralleled those made by me and will not be repeated. The distance of the sigmoid sinus from the external auditory canal was said to vary from 1 to 25 mm., and the statement was made that the right sinus was usually farther forward and larger than the left, but no actual measurements were quoted on this. I found that the size of the sinus groove always approximated that of the venous outlet of the jugular foramen. As the jugular foramen could be

TABLE 2

	Right			Left		
	Condyloid Foramen	Mastoid Emissary	Jugular Foramen	Condyloid Foramen	Mastoid Emissary	Jugular Foramen
Absent	39 (26%)	48 (33%)		47 (32.4%)	46 (31.7%)	
Present	106	97		98	99	
Average size (mm.)	2.5	1.55	7.2	2.4	1.54	6.5
Largest (mm.)	5	3.5	11	7	5	10

TABLE 3

	Right	Left
Distance of sigmoid sinus from external auditory canal	Average 11.0 mm.	Average 12.07 mm.
Farthest back	16 mm.	18 mm.
Farthest forward	Dehiscence into external canal	Dehiscence into external canal

measured more accurately than the width of the sinus groove, the size of the jugular foramen was used as an index of comparison of the size of the right and the left sigmoid sinus. Table 2 is a general summary of the measurements on 145 skulls.

The smallest jugular foramen on the right side was 2 mm. There was no sigmoid sinus groove and the groove for the inferior petrosal sinus was much larger than the average. A similar condition was found on the left side of another skull, the left jugular foramen being 3 mm. and the right one 6 mm.

10. Streit, in Braun, A.: Sinus Thrombophlebitis, New York, Paul B. Hoeber, Inc., 1928.

11. Denker, A., and Kahler, O.: Die Krankheiten des Gehörorgans, in Handbuch der Hals-Nasen-Orhen-Heilkunde mit Einschluss der Grenzgebiete, Berlin, Julius Springer, 1926, vol. 8.

A study of the measurements on each skull to determine if the position of the sinus gave any indication of the presence or absence of collateral circulation showed that there was no relationship between the position of the sinus and the amount of collateral circulation, as estimated by the size of the associated emissary foramina. Nor was there any relation between the size of the jugular foramen and the size of the emissary foramina.

In 55 skulls the size of the jugular foramen and the position of the sinus on the right and the left side were compared. In 19 instances the left and in 24 instances the right jugular foramen was the larger (table 3). In the rest there was 1 mm. or less difference. In 19 the left sinus was farther forward than the right, and in 20 the right was farther forward than the left. The others showed 1 mm. or less difference. The greatest difference in position of the two sinuses in 1 skull was 4 mm. Several showed 3 mm. difference and the rest only 2 mm. This study casts some doubt on the generally accepted statement that the right sinus tends to be farther forward, and much larger than the left one.

The jugular foramina were within the general average in both the anterior and the posterior group of sinuses. Hence, it would seem that the size of the sinus does not influence its position in relation to the external auditory meatus.

SUMMARY

The salient features of 21 consecutive cases of otitic sepsis are discussed.

A definite chill preceded the elevation in temperature in all adults but was absent in patients below 10 years of age. One third of the patients between 10 and 15 years of age had chills.

Culture of the blood gave positive results in 11 cases.

Absence of leukocytosis in a few instances was noted.

While the series of cases presented is too small to warrant any definite conclusions as to the value of ligation of the internal jugular vein in the treatment of otitic sepsis, this procedure seemed to be the least essential one in the management of the condition.

The mortality in these cases was 24 per cent.

An anatomic study of the sigmoid sinus is presented.

LYSOZYME OF NASAL MUCUS

METHOD OF PREPARATION AND PRELIMINARY REPORT ON ITS EFFECT ON GROWTH AND VIRULENCE OF COMMON PATHOGENS OF PARANASAL SINUSES

SYLVESTER DALY, M.D.
NEW YORK

Lysozyme (lytic enzyme) as first reported and described by Fleming [1] in 1922 is a substance found in various body tissues and secretions which has the property of dissolving certain bacteria. Since Fleming's initial work, lysozyme has been studied by many different investigators. Wolff [2] has reported on its chemistry, Hoder [3] on its relation to immunology, Anderson [4] on its importance in vitamin A deficiency, Corper [5] on its relation to tuberculosis, Meyer, Thompson, Palmer and Khorazo [6] on its purification and properties and Meyer [7] and his associates on the mechanism of its action. Fleming [8] in 1929 published a comprehensive review of the facts known up to that time.

ACTION OF LYSOZYME ON PATHOGENIC ORGANISMS

In his review Fleming [8] stressed the fact that while lysozyme is most active against nonpathogenic bacteria, it can attack pathogenic organisms when allowed to act in the full strength in which it occurs in some parts of the body. He showed that lysozyme from different

From the Departments of Oto-Laryngology and Bacteriology, College of Physicians and Surgeons, Presbyterian Hospital, Columbia University.

1. Fleming, A.: On a Remarkable Bacteriolytic Ferment Found in Tissues and Secretions, Proc. Roy. Soc., London, s.B **93**:306, 1922.

2. Wolff, L. K.: Untersuchungen ueber das Lysozym, Ztschr. f. Immunitätsforsch. u. exper. Therap. **50**:88, 1927.

3. Hoder, T.: Does Bacteriolytic Power of Lysozyme Bear Any Relation to the Action of Immune Bodies? Ztschr. f. Immunitätsforsch. u. exper. Therap. **70**:457, 1931.

4. Anderson, Oluf: Om lysozymets forhold ved xeropthalmi, Hospitalstid. **75**:1029, 1932.

5. Corper, H. J.: Lysozyme and Tuberculosis, Am. Rev. Tuberc. **25**:59, 1932.

6. Meyer, Karl; Thompson, Richard; Palmer, John W., and Khorazo, Devorah: Purification and Properties of Lysozyme, J. Biol. Chem. **113**:303, 1936.

7. Meyer, Karl, and others: Mechanism of Lysozyme Action, J. Biol. Chem. **113**:479, 1936.

8. Fleming, A.: Lysozyme: A Bacteriolytic Ferment Found Normally in Tissues and Secretions, Lancet **1**:217, 1929.

sources affects different organisms: Pneumococcus was attacked only by the lysozyme of pus and Streptococcus haemolyticus only by the lysozyme of tears. Staphylococcus and Streptococcus viridans were more affected by the lysozyme of nasal mucus than by that of tears or of pus. Thompson and Khorazo,[9] working with egg white lysozyme (which may act differently from lysozyme derived from body tissues), found that none of the ninety-nine strains of Staphylococcus albus and Staphyococcus aureus tested by them showed marked susceptibility to the lytic action of the lysozyme. Thompson, in unpublished work, stated that he could not demonstrate that egg white lysozyme had any effect on vaccine virus.

POSSIBLE RELATION OF LYSOZYME TO OTHER BACTERICIDAL SUBSTANCES IN NASAL SECRETION

The presence of a bactericidal substance active against Corynebacterium diphtheriae, Bacillus subtilis and Bacillus anthracis in normal nasal mucus was demonstrated by Ignatius.[10] Its properties resemble those of "inhibin," the bacteriostatic enzymic factor in saliva described by Dold[11] and by Weigmann and Koehn.[12] The relation of these local, nonserologic immune substances to lysozyme is not at present understood.

This article is a report of a method of preparation of lysozyme from nasal mucus and of preliminary studies of the effect of this lysozyme on common pathogens of the paranasal sinuses. A study of this type seemed desirable because most of the previous work has been done on lysozyme obtained from sources other than nasal mucus. Those workers who have studied nasal lysozyme (Fleming[1] and Hilding[13]) have obtained their material by irrigation of the noses of subjects suffering from coryza. Material obtained in this way is almost certain to contain many leukocytes which themselves contain lysozyme. For the purpose of studying the lysozyme of normal nasal mucus, the method to be described was devised.

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9. Thompson, Richard, and Khorazo, Devorah: Susceptibility to Lysozyme of Staphylococci, Proc. Soc. Exper. Biol. & Med. **33**:299, 1935.

10. Ignatius, A.: Ueber antibakterielle Hemmungsstoffe (Inhibine) im schleimigen Nasensekret, Ztschr. f. Hyg. u. Infektionskr. **118**:445, 1936.

11. Dold, Hermann: Beiträge zur Epidemiologie der Diphtheria, Zentralbl. f. Bakt. **135**:69, 1935.

12. Weigmann, F., and Koehn, A.: Weitere Untersuchungen über die Wirkung des menschlichen Speichels auf Diphtheriebacillen; die entwicklungshemmende bis keimtötende Wirkung des Speichels auf die Diphtheriebacilleatypen, Ztschr. f. Hyg. u. Infectionskr. **118**:507, 1936.

13. Hilding, Anderson: Changes in Lysozyme Content of Nasal Mucus During Colds, Arch. Otolaryng. **20**:38 (July) 1934.

METHOD OF PREPARATION OF LYSOZYME FROM NASAL MUCUS

In order to test the effect of lysozyme on bacterial growth a sterile preparation was required. The method used in obtaining this preparation consisted essentially in extracting dried, acetone-precipitated nasal mucus with dilute acid and sterilizing this extract by filtration. Meyer, Thompson and their associates [6] have shown that lysozyme may be readily filered (through a Berkefeld filter) when in acid solution.

Nasal mucus was obtained from a group of patients presenting themselves at the Vanderbilt Clinic for tonsillectomy. These patients, approximately 100 in number, were selected because they had no head cold, sinusitis, or purulent nasal condition. The nasal mucosa was apparently normal, and the material secured was normal nasal mucus. No patient with turbinal edema, a purulent exudate or other detectable evidence of intranasal infection was included. A pledget of sterile cotton soaked in sterile water was placed in close contact with the mucosa of both the inferior and the middle turbinates so that it extended posteriorly into the nasopharynx. This cotton was left in situ for ten minutes, to stimulate the secretion of nasal mucus. After ten minutes the cotton was removed and the thick, glairy nasal secretion was withdrawn through a long suction tip into a specimen bottle containing acetone. The mucus was left in the acetone for from twenty-four to forty-eight hours, to ensure thorough precipitation of the lysozyme. The collections of mucus from different patients taken on different days were pooled in an Erlenmeyer flask containing acetone.

The acetone liquid containing the precipitated lysozyme was centrifuged in pointed centrifuge tubes at 2,000 revolutions per minute for one-half hour. The supernatant acetone was decanted, and the mucus residue was dried in vacuo in a heated desiccator over calcium chloride. The dried material was weighed, placed in a sterile mortar and ground to a fine powder. The powdered mucus was then suspended in sterile saline solutions by addition of 5 to 10 cc. of saline solution to the mortar. The amount of saline solution added depended on the weight of the dried mucus, an effort being made to keep the volume as small as possible. The saline suspension was then acidulated by addition of sterile, boiled tenth-normal and then twentieth-normal hydrochloric acid, drop by drop until the suspension was acid first to litmus and then to congo red. The acidified saline material was then filtered through a sterile Berkefeld V filter, the filter having been previously acidulated with hundredth-normal hydrochloric acid. To the sterile acid filtrate boiled tenth-normal and later twentieth-normal sodium hydroxide were added drop by drop until a slight clouding appeared. At this point the filtrate was still slightly acid to litmus. Further neutralization caused precipitation of the lysozyme.

TECHNIC OF TESTING STRENGTH OF LYSOZYME PREPARATIONS

A typical strain of Sarcina (an air coccus), isolated from the air of the laboratory and carried on agar slants, was used as the test organism. In routine tests to determine the titer of the lysozyme from a specimen of nasal mucus, twenty-four hour old cultures of Sarcina on agar slants were suspended in saline solution to produce a turbidity equivalent to that of a no. 8 barium sulfate standard. In a series of small test tubes five-tenths cubic centimeter each of progressive dilutions of the lysozyme preparation was added to an equal quantity of the bacterial suspension. The tubes were incubated in a water bath at 37 C. for one-half hour and then placed in the ice box overnight.

TABLE 1.—*Illustrative Lysozyme Titration of Pooled Nasal Mucus Demonstrated by the Lysis of Standard Sarcina Suspension By Various Concentrations of Mucus Extract* *

Tube	Mg. of Dried Mucus per Cc. of Final Mixture	Degree of Lysis
1	5.6	4
2	2.8	4
3	1.4	4
4	0.7	4
5	0.35	3
6	0.175	3
7	0.087	2
8	0.43	2
9	0.21	1
10	0.10	1
Control	0	0

* Weight of dried mucus 112 mg. and volume of extract approximately 10 cc.—11.2 mg. of dried mucus per cubic centimeter.

The particular sample of pooled nasal mucus used in this illustrative titration was the most potent obtained in the course of this work.

Readings were made at the end of the half-hour and of the twenty-four hour period. These readings were made by determining the highest dilution giving complete lysis of the bacteria, as shown by the water-like clarity of the tube's contents. Reading was made on the basis of 4, meaning complete lysis with water-like clarity; 3, not quite complete lysis; 2, moderate turbidity; 1, heavy turbidity with only slight lysis, and 0, no appreciable lysis as compared with control.

EFFECT OF NASAL LYSOZYME ON GROWTH OF PATHOGENIC COCCI

Since most workers agree that lysozyme exerts little if any action on pathogenic cocci, it was considered important to utilize a method which would detect the slightest antibacterial activity. It seemed likely that such slight activity would be demonstrated best by the inhibition of growth in a liquid medium.

Extracts of nasal lysozyme prepared in the manner described were added to tubes of buffered infusion broth (p_H 7.5) to obtain the final concentration, shown in the tables of the experiments (tables 2, 3 and 4). Heavy inoculations of the organisms to be tested were made into the mixtures and into control broth containing no lysozyme. The tubes in all experiments were incubated for twenty-four hours at 37 C. The organisms used were Staph. aureus (a hemolytic strain), Pneumococcus type III and Str. haemolyticus, all recently isolated from material from sinus infections and maintained by frequent transfers in blood broth.

The results of these experiments are given in tables 2, 3 and 4.

TABLE 2.—*Effect of Nasal Lysozyme on the Growth of Pathogenic Cocci—Experiment 1* *

Organism	Final Concentration (Mg. of Dried Mucus per Cc. of Final Mixture)	Effect on Growth in Broth Culture
Pneumococcus type III	1.1	Uniform growth; evenly cloudy
	0.55	Uniform growth; evenly cloudy
	0 (control)	Uniform growth; evenly cloudy
Str. haemolyticus	1.1	Uniform growth; evenly cloudy
	0.55	Uniform growth; evenly cloudy
	0 (control)	Uniform growth; evenly cloudy
Staph. aureus haemolyticus	1.1	Growth in granular clumps
	0.55	Growth in granular clumps
	0 (control)	Uniform growth; no clumps

* This sample of nasal mucus when routinely tested for lysozyme activity by lysis of a standard suspension of Sarcina gave grade 3 lysis in a concentration of 0.55 mg. per cubic centimeter and grade 1 lysis in a concentration of 0.14 mg. per cubic centimeter.

TABLE 3.—*Effect of Nasal Lysozyme on the Growth of Pathogenic Cocci—Experiment 2* *

Organism	Final Concentration (Mg. of Dried Mucus per Cc. of Final Mixture)	Effect on Growth in Broth Culture
Pneumococcus type III	7.3	Clumpy, agglutinative growth
	2.4	Clumpy, agglutinative growth
	0 (control)	Evenly cloudy; uniform growth
Str. haemolyticus	7.3	Clumpy, agglutinative growth
	2.4	Clumpy, agglutinative growth
	0 (control)	Evenly cloudy when shaken
Staph. aureus haemolyticus	7.3	Clumpy, agglutinative growth
	2.4	Clumpy, agglutinative growth
	0 (control)	Evenly cloudy when shaken

* This sample of nasal mucus when tested with Sarcina gave grade 4 lysis in a concentration of 0.6 mg. and grade 1 lysis in a concentration of 0.04 mg. per cubic centimeter.

Blood plates streaked from lysozyme tubes and from control tubes in all cases showed good and apparently equal growths of the various organisms. The agar pour plate count of the granular growths in the staphylococcus broth and that of growths in the control broth were considerably different, the lysozyme broth containing only 107,000,000 staphylococci per cubic centimeter, in contrast to the 1,410,000,000 per cubic centimeter contained in the control broth. To what extent this difference in count was due to clumping of bacteria and to what extent it was due to actual inhibition of growth it is difficult to say.

TABLE 4.—*Effect of Nasal Lysozyme on the Growth of Pathogenic Cocci—Experiment 3**

Organism	Final Concentration (Mg. of Dried Mucus per Cc. of Final Mixture)	Effect on Growth in Broth Culture
Pneumococcus type III	9.0	Granular, flocculent growth
	0 (control)	Evenly cloudy when shaken
Str. haemolyticus	9.0	Granular, flocculent growth
	0 (control)	Evenly cloudy when shaken
Staph. aureus haemolyticus	9.0	Granular, flocculent growth
	0 (control)	Evenly cloudy when shaken

* This sample of nasal mucus when tested with Sarcina gave grade 4 lysis in a concentration of 0.12 mg. and grade 2 lysis in a concentration of 0.007 mg.

TABLE 5.—*Effect of Nasal Lysozyme on the Virulence of Pneumococcus Type III—Experiments 1 and 2**

No. of Mice	Dilution of Broth Culture† Mixed with Lysozyme or Saline Solution	Amount of Final Mixture Injected	Results	
			Experiment 1‡	Experiment 2§
2	Undiluted culture and lysozyme	0.5 cc.	Both mice died within 36 hours; pneumococcus type III recovered from peritoneum	Both mice died within 24-36 hours; pneumococcus type III recovered from peritoneum and heart blood
2 (controls)	Undiluted culture and saline solution	0.5 cc.	Both mice died within 24 hours; pneumococcus type III recovered from peritoneum and heart blood	Both mice died within 24-36 hours; pneumococcus type III recovered from peritoneum and heart blood
2	1:2 dilution and lysozyme	0.5 cc.	No fatality; both mice alive and apparently well 10 days after injection	Both mice died within 24-36 hours; pneumococcus type III recovered from peritoneum and heart blood
2 (controls)	1:2 dilution and saline solution	0.5 cc.	Both mice died within 24 hours; pneumococcus type III recovered from peritoneum and heart blood	Both mice died within 36 hours; pneumococcus type III recovered from peritoneum and heart blood
2	1:4 dilution and lysozyme	0.5 cc.	Both mice living and apparently well 10 days later	Both mice died within 24-36 hours; pneumococcus type III recovered from peritoneum and heart blood
2 (controls)	1:4 dilution and saline solution	0.5 cc.	Both mice died within 24 hours; pneumococcus type III recovered from peritoneum and heart blood	Both mice died within 24-36 hours; pneumococcus type III recovered from peritoneum and heart blood

* These samples of lysozyme when tested for lytic strength with Sarcina gave grade 4 lysis in a concentration of 0.7 mg. per cubic centimeter.

† Equal amounts (0.5 cc.) of broth culture and lysozyme and of broth culture and saline solution were used to make up the final mixture.

‡ The strain of pneumococcus type III used in this experiment was isolated from a patient with purulent sinusitis.

§ The strain of pneumococcus type III used in this experiment was isolated from the peritoneal cavity of a mouse.

Gram stains of the growths in the various tubes showed no apparent change in structure or in staining reaction as a result of the growth with lysozyme. The single but striking apparent effect of the lysozyme on the growth of these organisms was the granular nature of the growth in the lysozyme tubes. In most cases the clumps of bacteria settled to the bottom of the test tube, leaving a clear supernatant fluid above. Staph. aureus showed this granular growth even in the lowest concentrations of lysozyme. Pneumococcus and Str. haemolyticus did not show the granular growth in the lower concentrations of the lysozyme but showed it in the higher concentrations.

Since the lysozyme preparations were left on the acid side of neutrality, it is possible that the granular growth was caused by this acidity. However, colorimetric determinations of the hydrogen ion concentration of the lysozyme mixtures and of the control broth never showed a difference of more than 0.3 p_H. It is not likely that this difference is sufficient to explain the granular growth in the lysozyme tubes.

EFFECT OF NASAL LYSOZYME ON VIRULENCE OF PNEUMOCOCCUS TYPE III

EXPERIMENT 1.—A strain of Pneumococcus type III isolated from a patient with purulent sinusitis was cultured in blood broth and carried for five months with weekly transplants on fresh broth. Small amounts (0.5 cc.) of a twenty-four hour old broth culture of this organism undiluted and in dilutions of 1:2 and 1:4 were placed in small test tubes with equal amounts (0.5 cc.) of nasal mucus lysozyme and incubated at 37 C. for one hour in a water bath. After incubation 0.5 cc. amounts of the three different concentrations of this culture-lysozyme mixture were injected into the respective peritoneal cavities of a series of white mice. The control mice received injections of a mixture of broth culture and saline solution without lysozyme. The results are shown in table 5.

EXPERIMENT 2.—With the same organism as that used in experiment 1 but recovered in this case from the peritoneum of a control mouse and with a fresh sample of lysozyme of equivalent concentration (grade 4 lysis in a concentration of 0.7 mg. per cubic centimeter), experiment 1 was repeated.

In experiment 1 the organism in the lower concentrations of the broth culture, those in the 1:2 and in the 1:4 dilutions, were apparently rendered avirulent owing to the action of the lysozyme. However, organisms of the same strain after mouse passage were not affected by equivalent concentrations of lysozyme.

SUMMARY AND COMMENT

The presence of lysozyme in normal nasal mucus was demonstrated.

A method for obtaining sterile preparations of lysozyme from nasal mucus is described.

Nasal lysozyme in sufficient concentrations caused Staph. aureus, Str. haemolyticus and Pneumococcus type III to grow in a granular manner. Inhibition of growth may have occurred, but the reduced number of colonies obtained by pour plate methods from broth culture tubes containing lysozyme may have been caused by the clumping of organisms.

In one experiment Pneumococcus type III apparently suffered some loss of virulence as a result of contact with nasal lysozyme. However, a subsequent experiment showed that the same strain after mouse passage (which presumably increased its virulence) was not affected by equivalent concentrations of lysozyme. The results of these experiments on Pneumococcus type III are inconclusive, but they seem worthy of note, since they are in agreement with the findings of Thompson and Khorazo.[14] These workers had similar results with egg white lysozyme, which apparently destroyed the virulence of one strain of Pneumococcus but did not affect more virulent strains.

14. Thompson, R., and Khorazo, D.: Personal communication to the author.

LOCAL ANESTHESIA FOR SURGICAL TREATMENT OF THE SINUSES

FREDERICK T. HILL, M.D.
WATERVILLE, MAINE

The superiority of local over general anesthesia in surgical treatment of the sinuses is obvious, provided satisfactory anesthesia is obtained. The best index of satisfactory anesthesia is the reaction of the patient. It is well worth while to spend sufficient time and effort to obtain freedom from pain and to mitigate in every way possible the unpleasant features incidental to any operative procedure. More careful attention to technical details and preoperative preparation of the patient with barbiturates has accomplished a great deal in this respect. Any minor addition, however trivial, to the efficacy of the anesthesia would appear worthy of consideration. No claim is made to originality in the technic described. In conversations with numerous physicians throughout the country, however, I have found many who were not conversant with it.

For the external fronto-ethmoid operation local anesthesia according to the technic described by Sewall and others has been satisfactory in the main, although for this procedure, bound to be tedious and more or less terrifying to the patient, I have found that avertin (tribromethanol) reenforced by local anesthesia seems to be even more satisfactory from the patient's point of view. This opinion is based on information secured from several patients who have had this operation performed on the two sides at different times and with different forms of anesthesia. For the Caldwell-Luc procedure the usual technic, of injection of procaine hydrochloride in the region of the posterior superior dental and of the infra-orbital nerve plus application of cocaine intranasally, has likewise been satisfactory, except that removal of the antral lining was apt to be painful unless the interior was packed with cocaine-moistened strips of gauze for a sufficient time.

While cocainization of the nasal cavity usually has given sufficient anesthesia for the operator to perform an intranasal ethmoidectomy, it was a rare case in which the anesthesia, from the patient's point of view, could not have been improved on. Usually it was only partial. In my experience the most unsatisfactory results with local anesthesia have been in what is probably the simplest of sinal operations, intranasal

Read before the New England Otological and Laryngological Society, Nov. 18, 1936.

antrotomy. Despite all possible care as to the application of cocaine, unless the patient was so depressed by preliminary medication that the effect was practically that of a general anesthetic, there was considerable discomfort. The Caldwell-Luc technic gave less operative pain than the simple antrotomy. In making this statement I assume that a large window, extending practically the whole depth of the inferior meatus and well to the floor of the nose, would be the operative objective.

In considerably over three hundred such intranasal antral operations, complete and perfectly satisfactory anesthesia, from the patient's point

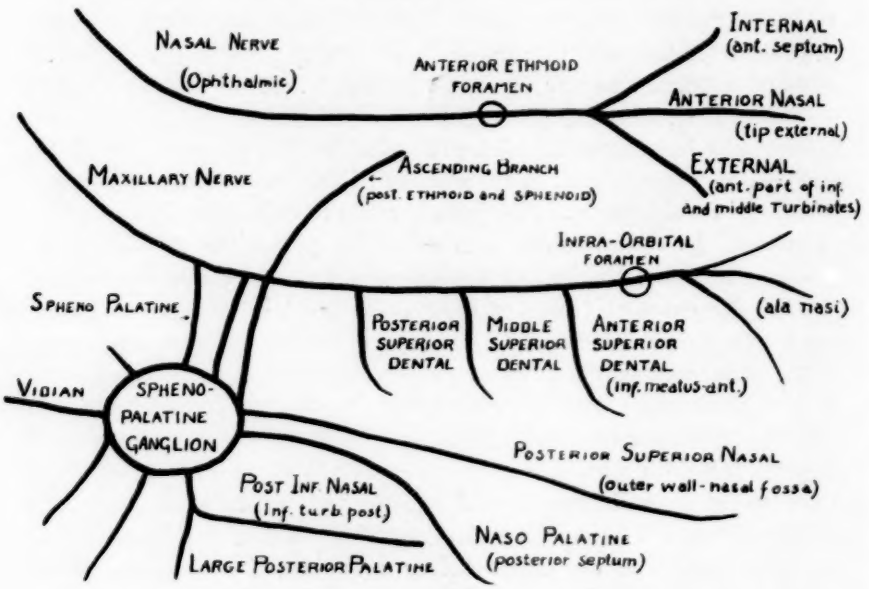

Fig. 1.—Diagram showing the nasal distribution of the maxillary and the nasal nerve (modified from Piersol, G. A.: Human Anatomy, Philadelphia, J. B. Lippincott Company, 1906, p. 1238).

of view, has been obtained by a direct deep injection into the infra-orbital foramen, plus the usual cocainization of the inferior meatus. An ordinary 22 gage hypodermic needle is employed. After palpation of the infra-orbital foramen to ascertain its size and direction, which may vary considerably, the needle is inserted directly through the skin and a few drops of a 2 per cent solution of procaine hydrochloride injected, superficially at first and then more deeply. After a minute or so the needle is advanced into the foramen to its full length and locked in by pressure of the first and the second finger of the left hand

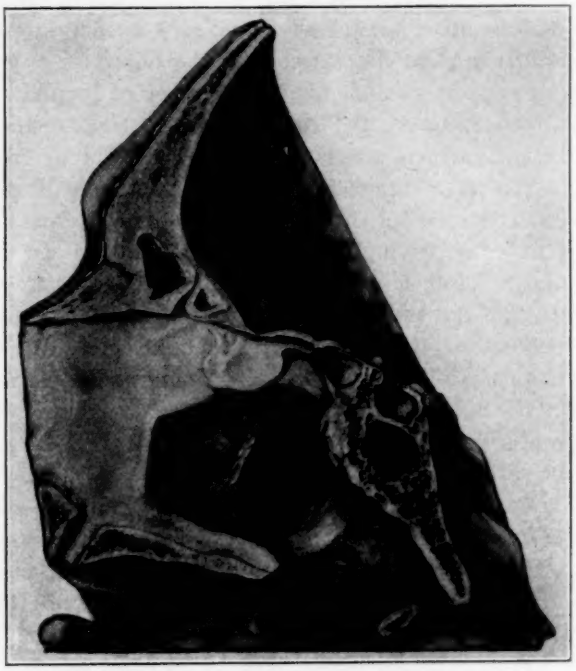

Fig. 2.—Dissection of the head of a cadaver, showing staining of Meckel's ganglion from injection of dye into the infra-orbital foramen with methylene blue.

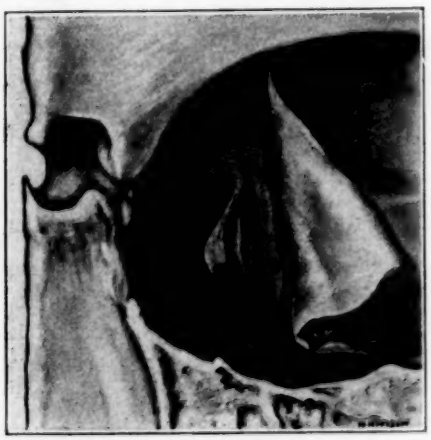

Fig. 3.—Dissection showing staining of the gasserian ganglion from injection of dye into the infra-orbital foramen.

straddling the butt of the needle, and from 2 to 2.5 cc. of the solution of procaine hydrochloride is slowly injected, the operator taking about five minutes to complete the injection. The object is to infiltrate the solution as far back as possible along the course of the maxillary nerve, blocking off the anterior, the middle and posterior superior dental nerve and the two or three sphenopalatine nerves running from Meckel's ganglion. If this is accomplished, there is blockage of the posterior superior nasal nerve, supplying the outer wall of the nasal fossa; the nasopalatine nerve supplying the posterior portion of the septum; the posterior inferior nasal nerve, supplying the posterior part of the inferior turbinate, and the ascending branch from Meckel's ganglion, going to the posterior ethmoid cells and the sphenoid sinus. In addition, the anesthesia of the middle terminal branch, going to the ala nasi, renders the insertion of the nasal speculum and the occasional stretching of the vestibule less disagreeable.

The same procedure is used in the Caldwell-Luc operation, the customary injections underneath the lip being omitted except for a few drops along the intended line of the incision to lessen bleeding. Of course, this will not block off the branches of the nasal nerve which come from the ophthalmic division of the fifth nerve. The external branch of the nasal nerve, which supplies the anterior part of the middle and the inferior turbinate, can be blocked off at the anterior ethmoid foramen by injecting along the nasal wall of the orbit, if one contemplates attacking the ethmoid sinuses transantrally in the Caldwell-Luc operation. If no extended removal of the ethmoid bone is to be done, cocainization of the middle turbinate on both the lateral and the mesial aspect will usually suffice. For intranasal ethmoidectomy this infra-orbital injection, plus orbital infiltration at the anterior ethmoid foramen and the usual intranasal cocainization, gives perfectly satisfactory anesthesia. It seems to be worth while to add the infra-orbital injection to the routine technic for the external fronto-ethmoid operation.

Injection of methylene blue into the infra-orbital foramen of the head of a cadaver after employment of this technic showed the dye infiltrating the sphenopalatine ganglion and its palatine branches. There was no infiltration of the orbit, the dye remaining inside the sheath of the nerve even after leaving the bony canal. In the cadaver, at least, there was penetration of the dye to the gasserian ganglion.

RECURRENT PARALYSIS OF THE LARYNX FOLLOWING INJECTION OF TETANUS ANTITOXIN

REPORT OF A CASE

A. HARRY NEFFSON, M.D.
NEW YORK

Paralysis following the injection of antitoxin, although rare in comparison with the frequency of serotherapy, has been reported in nearly a hundred cases since it was first noted, in the early part of the century, by Gangolphe.[1] In most of these cases paralysis has followed the injection of tetanus antitoxin, but other serums, such as those used in the treatment of diphtheria, scarlatina, pneumonia and meningococcic meningitis, have been implicated. Doyle,[2] in a tabulation of forty-nine cases of neurologic complications of serum disease, found involvement of the brachial plexus more frequent (thirty-three cases) than that of any other portion of the nervous system. Next in frequency was isolated involvement of the radial and the optic nerve; that of the latter was first described by Mason.[3] Cases of paralysis of the facial nerve, paralysis of the oculomotor nerve,[4] generalized paralysis [5] and involvement of the auditory nerve [6] have been reported. A search of the literature revealed three reports [7] from France and one [8] from Germany describing laryn-

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From the Laryngological Service of Dr. Rudolph Kramer at the Mount Sinai Hospital.

1. Gangolphe: Névrite au cours d'un cas de tétanos traité par la sérotherapie, Lyon méd. **110**:497, 1908.

2. Doyle, J. B.: Neurologic Complications of Serum Sickness, Am. J. M. Sc. **185**:484 (April) 1933.

3. Mason, V. K.: Optic Neuritis in Serum Sickness, J. A. M. A. **78**:88 (Jan. 14) 1922.

4. Valerio, A.: Oculomotor Paralysis After Preventive Injection of Tetanus Antitoxin, Arch. d'opht. **49**:181 (March) 1932.

5. Bourguignon, G.: Grave Generalized Paralysis with Bulbar and Medullary Symptoms Following Injection of Purified Antiserum, Rev. neurol. **1**:334 (March) 1931.

6. Cutter, R. D.: Auditory Nerve Involvement After Tetanus Antitoxin: First Reported Case, J. A. M. A. **106**:1006 (March 21) 1936.

7. (*a*) Lavrand, A.: Left Recurrent Paralysis Following Injection of Tetanus Antitoxin, J. d. sc. méd. de Lille **2**:331, 1924. (*b*) Roger, H.; Prévot, M., and Wahl, M.: Paralysis of Abductors of Larynx and of Velum Palati Following Prophylactic Injection of Antitetanus Serum, Rev. d'oto-neuro-opht. **12**:419 (June)

(Footnotes continued on next page)

geal paralysis following the injection of tetanus antitoxin. One of these [7b] was of bilateral palsy of the abductors, which remained practically unimproved after five months. The patient was then lost from sight. In the other three cases paralysis disappeared from two to five months after the onset. In all four cases paralysis appeared from six to nine days after the injection of serum. Besides the case reported by Imperatori,[9] there have been no reports of laryngeal paralysis following injection of serum in the American literature. Tetanus developed in Imperatori's patient, with trismus and generalized paralysis, and was treated with large doses of antitetanus serum. About three weeks after the last injection of serum, one week after the patient's discharge from the hospital, edema of both arytenoid cartilages and bilateral recurrent paralysis developed, necessitating tracheotomy. The right vocal cord eventually recovered, but on the left side perichondritis of the larynx resulted. At no time was there any generalized reaction to the serum.

In an excellent review of the subject Kraus and Chaney [10] stated:

> It seems justifiable to conclude that the neuropathologic changes associated with serum sickness are the same as those which appear elsewhere in the body and consist of a primary disorder of the blood vessels, causing nutritive impairment of the tissues of the nervous system and interfering temporarily as a rule with the activity of the nerve fibers and cells, but occasionally causing cell death and parenchymal necrosis. A vascular disorder also produces the meningeal picture and excess of spinal fluid. An edematous process may occur in the perineural sheaths, or the nutrition of the roots or nerves may be interfered with by impairment in the blood supply, thus causing various radicular and neural syndromes. . . . Serum sickness involving the nervous system produces a group of syndromes almost as varied and numerous as syphilis or epidemic encephalitis. . . . The syndromes may be divided into: (1) cerebral, (2) spinal, (3) radicular and (4) neural. . . . Various combinations of these syndromes can occur.

REPORT OF CASE

V. T., a 19 year old Italian youth, was admitted to the laryngologic service of Dr. Rudolph Kramer at the Mount Sinai Hospital on Nov. 14, 1936. Four weeks before admission his left foot had been injured with an ice pick, and that day 1,500 units of tetanus antitoxin had been injected into his left arm after sensi-

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1934. (*c*) Roger, H., and Bremond, M.: Right Unilateral Paralysis of Larynx Following Prophylactic Antitetanus Injection, ibid. **12**:423 (June) 1934.

8. Schauwecker, J.: Serum Sickness and Recurrent Paralysis Following Injection for Prevention of Traumatic Tetanus, Zentralbl. f. Chir. **60**:1411 (June 17) 1933.

9. Imperatori, C. J.: Bilateral Abductor Paralysis of Larynx Caused by Injection of Antiserum and Its Subsequent Stenosis Due to Perichondritis: Case Report, Tr. Am. Laryng. A. **57**:240, 1935.

10. Kraus, W. M., and Chaney, L. B.: Serum Disease of the Nervous System: Report of Three Cases, Arch. Neurol. & Psychiat. **37**:1035 (May) 1937.

tivity tests of the skin and the conjunctiva had given negative results. On the following day the site of the injection became swollen, red and painful. Two days later a generalized urticarial eruption appeared. On the third day the patient awoke with generalized muscular and articular pains, and in the afternoon he suddenly became hoarse. The hoarseness increased so rapidly that within a few hours he could "hardly talk." He stated that during the preceding two weeks his voice had improved gradually but that he was still hoarse. At no time was there noted any diplopia, nasal regurgitation, muscular weakness or other neurologic condition. His history was entirely irrelevant.

The essential physical findings at the time of admission follow: There was complete paralysis of the left vocal cord, with diminution of sensation on the left side of the larynx. The left pupil was slightly larger than the right. The reactions to light and in accommodation, the external ocular movements and the fundi were normal. There were an equivocal Babinski sign on the left and questionable paresis of the right side of the face of central origin. The neurologic examination showed no other abnormality. Blood count, urinalysis, roentgenographic study of the chest and the neck and Wassermann and the Kahn tests of the blood showed nothing abnormal. The temperature was normal. The neurologic staff felt that the picture suggested a serum reaction causing a localized hemorrhage, in the medulla, involving the left nucleus ambiguus. Perineural edema and impairment of the blood supply of the laryngeal nerve were considered less likely causative factors.

The laryngeal paralysis improved gradually and disappeared completely three months after the onset.

152 East Ninety-Fourth Street.

PHLEGMON AND FISTULA FROM CALCULUS OF THE SUBMAXILLARY GLAND

JOHN H. CHILDREY, M.D.
SAN FRANCISCO

Although not extremely rare, cases of submaxillary calculus with phlegmon and fistula are unusual enough to justify their being reported. The case described here is fairly typical of the more severe type.

REPORT OF A CASE

A man aged 45 came for examination on July 23, 1937, because of a painful swelling of two weeks' duration in the upper part of the right side of his neck,

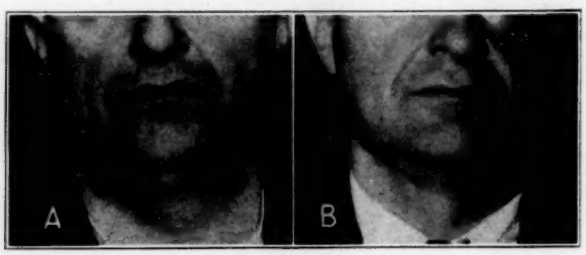

Fig. 1.—*A* shows the lower portion of the patient's face before operation, and *B* shows the same area after operation.

associated with a discharge of pus into the mouth. He stated that a half-pint (236.59 cc.) of pus had been expectorated at the onset of the attack.

Twenty years previously the right side of the floor of the mouth had been burned by a hot dental filling, and two months afterward he had noticed soreness in this region. Three years later a roentgenogram had shown a tiny shadow in the floor of the mouth on the right side. During the ten years preceding the examination he had had occasional attacks of pain and swelling in the right submaxillary gland, usually brought on by eating, particularly of rich foods. During the four years preceding the examination he had had frequent exacerbations of the inflammation characterized by painful swelling of the right side of the floor of the mouth and the upper part of the right side of the neck, associated with general malaise and a discharge of much pus into the mouth. This swelling had been drained by an operation performed elsewhere on three occasions, with temporary benefit, the incisions being made within the mouth.

The patient spoke thickly. There was a moderately tender irregular mass approximately 5 by 6 by 6 cm. in the right submaxillary region. There were

swelling of the tissues of the right hypopharynx and of the right side of the neck, redness of the right palate and tonsil and a small fistula situated posteriorly in the floor of the mouth. Palpation did not reveal a calculus.

A roentgenogram showed a shadow in the right submaxillary gland. The leukocytes numbered 20,640, of which 83 per cent were polymorphonuclear.

For the first days after the examination the infection was treated with hot applications to the neck and hot irrigations of the mouth. A considerable amount of pus drained from both the duct and the fistula, and by July 28 the inflammation had subsided sufficiently to warrant excision of the right submaxillary gland. This was done with the patient under ether anesthesia. The right external maxillary artery and the facial vein were ligated, and a rubber drain was left in the incision. The removed calculus measured 6 by 7 by 10 mm. Since the operation the patient has been complaining of dryness of the right side of the mouth, but he has had no recurrence of the other symptoms.

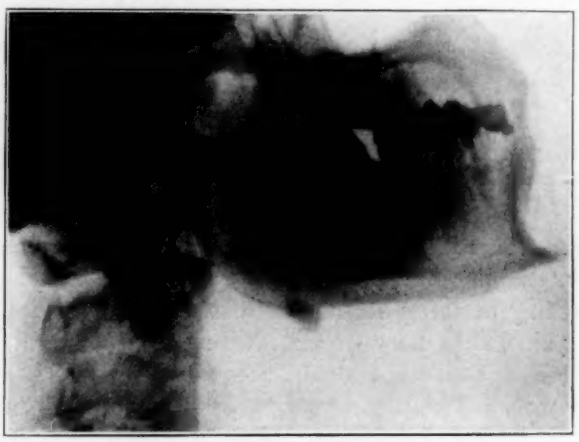

Fig. 2.—Roentgenogram showing a calculus in the gland.

COMMENT

This case is interesting in that the calculus had probably been present for twenty years and in that there were both a phlegmon and a fistula. Previous operations were inadequate. It is possible of course that if the stone alone had been removed the infection would not have recurred, but this is debatable. As pointed out by New & Harper [1] and by Ivy & Curtis,[2] whose series of cases are the largest that have been reported, the removal of the entire gland is the advisable treatment if there is considerable infection in the gland and the patient has a long history of recurring suppuration.

1. New, G. B., and Harper, F. R.: Chronic Inflammation of the Salivary Glands With or Without Calculi, Surg., Gynec. & Obst. **53**:456-460 (Oct.) 1931.

2. Ivy, R. H., and Curtis, L.: Salivary Calculi, Ann. Surg. **96**:979-986 (Dec.) 1932.

Salivary calculi are found most often in the submaxillary gland or duct and less often in the sublingual gland. More than one gland may be affected, and several stones may be present. The right submaxillary gland seems to be more often affected than the left, and the condition is more common in males than in females. It usually occurs in middle life.

Various theories have been offered in an attempt to explain the formation of a calculus in the submaxillary gland. Some say that the calcium salts are precipitated by bacteria, by actinomycetes or as a result of stagnation of saliva or chronic inflammation of the duct. Others say that the calculus represents a congenital or developmental phenomenon and that the saliva from the submaxillary gland differs in chemical composition from that from the other salivary glands, which accounts for the formation of a calculus much as for that of tartar on the teeth. By roentgenographic and microscopic examination Berger and Berke[3] found in every case a focus about which the mineral salts were deposited. These foci appeared to be composed of inflammatory cells, of bacteria in several instances and of a basic substance which they believed to be primarily fibrinous and derived from an inflammatory exudate.

The most common complaint is of the repeated exacerbations of acute inflammation which result from irritation or obstruction due to a calculus in the gland or duct. During one of these attacks there is a tender swelling of the gland, usually brought on by eating and associated with pain in the floor of the mouth and in adjacent structures. Fever and leukocytosis are usually present. The duct itself may be tender and swollen, and it is usually possible to express pus from it. The pain may be mild or severe. After an acute flare-up there may be subsidence of symptoms for variable periods, of months or years, during which there may be only slight enlargement of the gland, little or no discomfort and little or no purulent discharge from the duct.

As a result of obstruction from a stone there may be congestion of the mucosa with erosion or ulceration; then granulations may form, followed by fibrosis and atrophy of the tissues of the gland. Infection commonly follows obstruction, and if it is marked there may be a phlegmon in the gland and the surrounding tissues; a fistula is occasionally one of the sequelae. The fistula may open into the mouth or on the skin and infrequently provides for spontaneous discharge of the calculus.

Diagnosis is not always easy, particularly in cases in which there are small stones or slight symptoms and mild attacks of submaxillary swelling. It is often possible to express pus from the submaxillary duct,

3. Berger, A., and Berke, J. D.: Calculus of the Submaxillary Gland, J. Am. Dent. A. **23**:1397-1418 (Aug.) 1936.

the opening of which may be reddened or swollen. On bimanual palpation one may feel a calculus in the duct or gland, unless it is too deeply situated in the latter. When the stone is situated within the gland, the making of a lateral extraoral roentgenogram is advisable. A calculus in the duct may be demonstrated by having the patient hold a no. 2 film between the teeth and directing the rays upward from under the chin. Roentgenograms should be made in every case, for use in differential diagnosis and because there may be multiple stones. One must differentiate between calculus and cellulitis originating in teeth or tonsils and also between calculus and malignant growth, actinomycosis or tuberculous or syphilitic swelling.

The patient usually presents himself for examination during an acute flare-up of the infection. This condition is best treated by hot irrigations and hot applications. Probing is contraindicated. When the acute condition subsides the stone is located, and if it is situated in the duct or anteriorly in the gland it may be removed through the mouth. This is not always easy, for it may be difficult to locate and fixate a small calculus. Removal of the stone may not result in subsidence of the infection because the gland may be considerably diseased, in which case its removal is advisable.

If the gland is removed, care is necessary to avoid injury to the lingual and hypoglossal nerves and to the inframandibular branch of the facial nerve. In the case reported here postoperative scarring was negligible.

Progress in Otolaryngology

Summaries of the Bibliographic Material Available in the Field of Otolaryngology

ANATOMY AND PHYSIOLOGY OF THE EAR

PHILIP E. MELTZER, M.D.

M. H. LURIE, M.D.

WERNER MUELLER, M.D.

BOSTON

This review of the literature dealing [1] with progress made in studies of the anatomic and physiologic relations of the ear does not cover all the available bibliographic material. We have utilized abstracts whenever possible, and translations were made when the articles were of such importance as to warrant the effort. The number of articles reviewed, therefore, is not great, but, in our opinion, is adequate to summarize the present day view.

ANATOMIC AND HISTOLOGIC RELATIONS OF THE LABYRINTH

Bast [2] introduced the name utriculo-endolymphatic valve to designate a highly cellular connective tissue flap lined by columnar epithelium which guards the utricular opening into the endolymphatic canal. The valve is described as a fold of the posterior wall of the utricle and the endolymphatic canal at the point of junction of the two structures. Serial sections of twenty-five human fetal ears formed the basis for the study.

Bast suggested that the position of the valve indicates that the flow of endolymph is into the utricle. He expressed the opinion that when the flow is in the opposite direction slow movement of endolymph may be permitted and that the valve may function to prevent a sudden outflow of endolymph from the utricle. No physiologic experiments were introduced.

Anson and Nesselrod [3] and Anson and Wilson [4] state that until recently little was known of the structure and shape of the endo-

1. Dr. Edgar M. Holmes, Dr. John R. Richardson and Dr. John R. Frazee, associates in the graduate course in otology at the Harvard University Medical School, gave assistance in this study.

2. Bast, T. H.: The Utriculo-Endolymphatic Valve, Anat. Rec. **40**:61-66, 1928.

3. Anson, B. J., and Nesselrod, J. P.: Endolymphatic and Associated Ducts in Man, Arch. Otolaryng. **24**:127-140 (Aug.) 1936.

4. Anson, B. J., and Wilson, J. G.: Form and Structure of Endolymphatic and Associated Ducts in the Child, Anat. Rec. **65**:485-498, 1936.

lymphatic duct. Indeed, the work on this subject may be said to have started in 1928 when Bast first described the "cusp or valve" which in the human embryo "projects into the utriculus and guards the utricular opening into the utriculo-endolymphatic duct."

Anson and his co-workers used the Born wax plate method in reconstructing the shape of the endolymphatic duct from serial sections. Their work proves conclusively that the stylized figures of the endolymphatic and associated ducts available in textbooks are not only inadequate but misleading. From the utricular extremity to the dural end, the duct is dissimilar to the reedlike, undifferentiated tube portrayed in the diagrams that hitherto have given students their conception of the anatomic relations of this duct. Further, the utricular duct is not circular in cross sections, nor is its utricular orifice round. Instead, the slitlike character of the utricular communication with the endolymphatic duct has been observed to be a striking feature. This communication is bounded on one aspect by a fold or "valve" (in complete accordance with Bast) which anatomically at least is similar to other mechanisms in the body known to have a valvular function.

The several dilatations of the endolymphatic duct (see plates [3]) are observed to be constant features. The endolymphatic duct is considerably enlarged to form a sinus-like space medial to the utricular fold. As it proceeds distally within the vestibular aqueduct, the duct at first narrows and then expands into a second sinus-like space, the walls of which are deeply plicate. These pleats are composed of epithelium over a substratum of loose, vascular connective tissue. The rest of the duct is marked by a decided narrowing, and then a distal swelling in the region commonly termed the endolymphatic sac. Here the plications may be taller, but the underlying tissue is much less vascular than that in the region of the intermediate dilatation.

The extensive folding of epithelium, presenting a relatively great amount of surface area, with each of the rugosities containing a core of vascular connective tissue, leads Anson and his co-workers to agree with the interpretation of Guild that this structure may well be a resorptive mechanism for endolymph. On anatomic grounds, the intermediate dilatation should be the region of greatest physiologic activity.

Perlman and Lindsay [5] attempt to substantiate further the presence and function of a valvelike fold at the junction of the utricle and the utricular duct, described in 1928 by Bast as the utriculo-endolymphatic valve. The valve was observed in eight human and ten other mammalian ears. In three pathologic specimens the staining characteristics of the utricular contents differed from those of the utricular duct, endolymphatic duct and saccule. It is suggested that the valve may function

5. Perlman, H. B., and Lindsay, J. R.: Utriculo-Endolymphatic Valve, Arch. Otolaryng. **24**:68-75 (July) 1936.

to maintain the volume of the utricle and semicircular canals at a constant level. This may explain the absence of vestibular response during sudden changes of pressure in the middle ear, such as are produced by inflation, changes of pressure in the perilymph not being translated into fluid movements within the semicircular canals.

ANATOMIC OBSERVATIONS ON THE MIDDLE EAR

Yates [6] correlates his observations made during the microdissections of a number of decalcified temporal bones. Deviations in the course of the facial nerve are noted. The folds of the mucous membrane in the middle ear are described in detail, as are the ossicular attachments and the intrinsic muscles. Twenty photographic enlargements portray beautiful dissections.

In order to study the pathways of infection from the middle ear, Hodjakoff [7] injected a 1 per cent solution of trypan blue into the middle ear of rabbits and guinea pigs. The results were as follows: 1. There was no remarkable precipitation of the dye anywhere, especially during inflammation. 2. Spread occurred (*a*) along the bony fissures and the fibrous wall of the facial nerve, with precipitation in the histiocytes of the epineurium and diffusion in the nerve fibers; (*b*) to a lesser extent along the bony canaliculi of the pyramid (toward the scala), with superficial staining of the histiocytes and diffuse precipitation into the mucoperiosteal fibers, and (*c*) into the inner ear by way of the secondary tympanic membrane, through the connective tissue of which the dye diffused, staining some of the histiocytes. Besides, Hodjakoff observed precipitation in the macrophages of the perilymphatic spaces.

Miodonski [8] studied the microscopic structure of the ear drum along the lines previously described by Luscher, with similar results. For practical purposes, he found that microscopy is useful in determining whether the drum is thinned as the result of atrophy or of scar formation. If the vessels are arranged normally, the case is one of atrophy; if they are interrupted, it is one of scar.

PNEUMATIZATION OF THE TEMPORAL BONE

Wittmaack's original treatise on pneumatization of the temporal bone remained for years the theory accepted by the vast majority of otolaryngologists. Cheattle and Mouré disproved the theory in its entirety

6. Yates, A. Lowndes: The Anatomy of the Middle Ear, J. Laryng. & Otol. **51**:476-483, 1936.

7. Hodjakoff, N. D.: Ueber die Ausbreitung von Trypanblau bei der Einführung in das Mittelohr, abstracted, Zentralbl. f. Hals-, Nasen- u. Ohrenh. **29**: 216, 1937.

8. Miodonski, J.: Beitrag zur Mikroskopie des Trommelfelles, abstracted. Zentralbl. f. Hals-, Nasen- u. Ohrenh. **29**:4, 1937.

and expressed the belief that the compact mastoid was of constitutional type. Then followed other investigators, such as Steurer, Schwarz and Albrecht, who claimed that the inherited constitutional character of the tissues plays an important role in the type of pneumatization. It is agreed by all that infections during infancy and early life may alter the plan or pattern of pneumatization of the mastoid. The controversy continues—one worker believing that the important factor is the epithelium and subepithelial tissues; another, the character of the osseous tissue, and another, a combination of both. Unquestionably, each investigator has contributed something of value to the understanding of pneumatization of the temporal bone.

Wittmaack, in his latest contribution,[9] holds firmly to his original belief; I quote from his summary.

> My explanation of the conditions investigated by Schwarz in association with myxomatous lymphoid tissue of the middle ear as postmortem putrefaction phenomena has not been confuted. The theory of Albrecht and Schwarz that these changes are morphologically recognizable signs of a genotypical hereditary disposition is not proved. The assumption based on this concept of a purely genotypical origin of the various constitutional forms of the mucous membrane is also unproved. That opposite constitutional forms of the mucous membrane are sometimes to be seen in the right and left ears of the same person overthrows this hypothesis. The theory is also refuted by the fact that in uniovular twins contrasting forms of pneumatization of the processus mastoideus occur. The explanation of the origin of the various constitutional forms of the mucous membrane given in my theory of pneumatization retains its validity.

Rüedi[10] approaches the problem of the growth of the middle ear space in the light of the following questions: 1. What part do the various tissues take in forming the middle ear space? 2. What are the results of normal development at various ages between the fifth embryonic month and the eleventh year of life? 3. What is the role of otitis media neonatorum in these processes? Rüedi examined fifty-three vertically sectioned temporal bones from thirty-five patients with histologically normal ears whose ages ranged from the fifth embryonic month to the eleventh year of life. The first section describes the development of the temporal bone from the fifth to the seventh embryonic month, and the second, that from the seventh month to birth; the third section compares the temporal bones of ten new-born children, and the fourth deals with the development of the mastoid process in childhood. The following subjects are discussed: topography, with special emphasis

9. von Wittmaack, K.: Ueber die morphologischen Grundlagen der sogenannten Ausfalls—und Reizerscheinungen des Vorhofbogengangsapparates bei entzündlichen Labyrintherkrankungen, Acta oto-laryng. **25**:109-128, 1937.

10. Rüedi, Luzius: Die Mittelohrraumentwicklung vom 5. Embryonalmonat bis zum 10. Lebensjahr, abstracted, Zentralbl. f. Hals-, Nasen- u. Ohrenh. **29**:2, 1937.

on the labyrinth as a necessary factor in growth of the middle ear, and the histologic structure of the middle ear, including that of the bone, periosteum and mesenchyma and the activity of the epithelium.

In considering the development of the middle ear, Rüedi differentiates two phases, the first of which is concerned with formation of the bony cavity of the middle ear and the second with pneumatization. It is thought that the first phase is determined by development of the mesenchyma, for the bony space is the result of the arrangement of differentiated periosteal layers. Cell formation in the bone is independent of the marrow spaces, of which even the largest has no influence on the direction in which growth takes place. It is claimed also that in the second phase the growth of the epithelium depends on the mesenchyma, the epithelium merely lining the cavity without in any way influencing the growth of the middle ear chamber. (Apparently Rüedi did not take into consideration the most recent literature on the the development of this space.) Individual variations appear to be important as regards not only the laying down of the bone but the formation of bone and air spaces from the mesenchyma. The idiotypic variations in pneumatization of the mastoid process in the sense of Albrecht and Schwarz are said to be due to complicated growth processes which are determined by the unknown (constitutional) influences of the mesenchyma. The infantile otitis in the cases in which examination was made had no influence on the development of pneumatization. Rüedi finally calls attention to a communication of Eckert-Möbius, in which it is stated that the primary cause of pneumatization of the mastoid process and the facial bone is to be looked for in the bone itself.

Yamashita [11] carried out extensive measurements on one hundred temporal bones of Japanese patients with reference to pneumatization of the outer portion of the bone and its relationship to the size of the mastoid process, the depth of the sigmoid sinus and the level of the floor of the middle cranial fossa. He concluded that pneumatization influences these relationships markedly. The study tends to verify that which has previously been recognized and described by those who have studied the influence of pneumatization on the structure of the temporal bone.

Yamashita,[12] in an article based on anatomic studies, establishes six types of pneumatization of the temporal bone. In type 1 there is enormously extensive pneumatization, with regularity of size, arrange-

11. Yamashita, K.: Die quantitative statische Untersuchung über die pneumatischen Zellen des äusseren Teiles des Schläfenbeins, abstracted, Zentralbl. f. Hals-, Nasen- u. Ohrenh. **28**:250, 1937.

12. Yamashita, K.: Ueber den Strukturzustand des pneumatischen Systems des Schläfenbeins und den Pneumatisationstypus, abstracted, Zentralbl. f. Hals-, Nasen- u. Ohrenh. **28**:250, 1937.

ment and direction of the cells and thickness of the intracellular wall and boundary between the pneumatic and the nonpneumatic portion of the bone. The last relationships Yamashita terms the structural state of the temporal bone. In type 2 pneumatization is of normal or greater than normal extent, with a regular structural state. In type 3 pnuematization is of the same extent as in type 2, with an irregular structural state. In type 4 there are less extensive pneumatization and regular structure, and in type 5, less extensive pneumatization and irregular structure. In type 6 pneumatization is least extensive, the structure being almost always irregular and showing no extension of pneumatization. Yamashita believes, on the assumption that pneumatization and the structural state are the same on the two sides, that considerable aid can be derived from the roentgenologic picture and comparison of the healthy and the diseased side.

Schwarz [13] finds that in pneumatization of the middle ear spaces, as well as the sinuses of the nose, the hereditary factor is of importance. This has been shown by roentgenograms of the mastoid processes of twins. Recently, Steurer again pointed out that the considerable difference which appears at times in the pneumatization of the right and of the left ear in the same person cannot be explained on the basis of an idiotypic arrangement of the mucous membrane but that external influences (otitis in infancy) must operate. On the basis of this conception, pneumatization of the mastoid processes was studied in entire families. In a family with a homologous pair of twins, a dominant distribution of compact mastoid processes in both ears was shown, whereas pneumatization of medium grade occurred in only one instance. In the study of other family groups it was asked whether the process of pneumatization can be inhibited or stopped by otitis media. Schwarz studied twenty-two subjects who in their youth had suffered from acute or chronic otitis following scarlatina, especially those in whom the disease occurred before the fifth year of life. The observations showed that the slight pneumatization of the mastoid process after otitis complicating scarlatina can be explained on the basis of a dominant hereditary factor in the distribution of compact mastoid processes. The objection might be raised that in these families an accidental increase in infantile otitides may have simulated the dominant hereditary tendency. There would then have to be: susceptibility in the family; otherwise, the tendency to the disease would not have been shown so often. This conception is in accord with the fact that in the members of these families a marked increase in the incidence of scarlatina could be proved and that the

13. Schwarz, M.: Der Erbfaktor bei der Pneumatisation des Warzenfortsatzes. Röntgenologische Stammbaumuntersuchungen, abstracted, Zentralbl. f. Hals-, Nasen- u. Ohrenh. **28**:542-543, 1937.

morbidity rate for scarlatina was disproportionately higher than that found in the literature.

Okasaki [14] examined twenty-nine pairs of homologous twins by Schüller's method. According to the criteria of Siemans, nineteen pairs were uniovular twins and eleven pairs biovular. With regard to the extent of pneumatization, the arrangement of the cells, the cell walls and the like, 75 per cent of the uniovular twins were alike, as compared with 45 per cent of the biovular twins. The similarity of the four ears of one pair of uniovular twins was so great that they could not be told apart. Okasaki therefore lays stress on the hereditary process in the formation of the mastoid process.

PHYSIOLOGIC REACTIONS OF THE COCHLEA

In the literature for the past year on the physiologic behavior of the cochlea, the emphasis is still on the interpretation and significance of the phenomenon of Wever and Bray. There have developed two schools of thought with regard to the source of this phenomenon. One school states that it consists of two parts: (*a*) electrophysiologic activity of the organ of Corti (cochlear response), and (*b*) action currents of the cochlear nerve.

The other school states that the cochlear response of the Wever and Bray phenonemon does not arise from the organ of Corti but from polarization of the membranes.

In order to understand the two schools, certain physiologic facts must be understood. The terms "action current" and "nerve impulse" are synonymous and mean the transmission of a nerve impulse along the nerve fiber. This transmission is usually demonstrated by the detection of electrical changes, or action currents, as they pass along the nerve. It is not necessary to use electrical means for detection of passing of the impulse along the nerve. This can be done by the neuromuscular reflex. A muscle with its nerve is isolated; on stimulation of the distal end of the nerve mechanically, chemically or electrically, the muscle contracts, showing that an impulse has passed down the nerve. Thus, from the physiologic standpoint, the action current and the nerve impulse are the same. The impulse (the exact nature of which is not known) is transmitted along the nerve and produces reactions in the organ that is stimulated by it. To persons interested in the basic phenomenon we advise reading a book by Adrian.[15]

The electrophysiologic effect (cochlear response) is concerned with the electrical changes that occur in the cochlea while functioning and

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14. Okasaki, M.: Röntgenologische Studien über die Bedeutung der Erbfaktoren bei der Pneumatisation der Warzenfortsätze, abstracted, Zentralbl. f. Hals-, Nasen- u. Ohrenh. **29**:217, 1937.

15. Adrian, E. D.: Basis of Sensation, New York, W. W. Norton & Company, 1928.

not with the nerve impulses (action currents). This type of activity is best shown by the electrocardiogram, which is a record of changes in electric potentials (currents) in the heart muscles as they relax and contract. This electrical effect is much like a radio broadcast. It spreads and can be picked up from any part of the body if the poles are placed with the organ between them. The action currents of the nerves can be found only in, and not outside, the nerves. They may be compared to the messages carried by a telephone wire from a central station to the broadcasting station. Thus, the messages are strictly confined to the telephone wire, while at the broadcasting station they are transformed so as to be sent out in all directions.

One school claims that the cochlear effect shows what is going on in the organ of Corti, while the action currents of the cochlear nerve indicate that the impulses are traveling up the nerve to the cortical areas, to be interpreted by the brain.

One must remember that testing the cochlea by the Wever and Bray phenomenon does not indicate the true hearing present. True hearing can be tested only by the mental or physical responses of an animal or human being to sound effects. It is generally assumed that a normal end-organ or nerve mechanism will give a normal response. When either is abnormal there will be an abnormal response or none.

The school which claims that the Wever and Bray effect is the result of polarization of the cochlear membranes also postulates that it is not an indication of the true activity of the organ of Corti. The theory has been advanced by Eyster, Bast and Krasno [16] and Ashcroft, Hallpike and Rawdon-Smith.[17] If one accepts the polarization theory of the origin of the Wever and Bray effect, it is impossible to interpret the findings in terms of the pathologic changes in the organ of Corti.

With the acceptance of the theory that the organ of Corti is the origin of the Wever and Bray phenomenon, as postulated by Davis, Saul, Lurie, Guttman, Hughson and others, the interpretation of pathologic changes in the organ of Corti and their correlation with the Wever and Bray phenomenon are possible. Lurie [18] takes the stand that degeneration of the external hair cells can be interpreted as a loss of from 30 to 40 decibels with the Wever and Bray effect and that loss of internal and external hair cells means a loss of from 60 to 90 decibels, depending on the extent of degeneration of the organ of

16. Eyster, J. A. E.; Bast, T. H., and Krasno, M. R.: The Origin of Cochlear Potentials, Laryngoscope **47**:461-479, 1937.

17. Ashcroft, D. W.; Hallpike, C. S., and Rawdon-Smith, A. F.: On the Changes in Histological Structure and Electrical Response of the Cochlea of the Cat Following Section of the VIIIIth Nerve, Proc. Roy. Soc., London, s.B **122**: 186-197, 1937.

18. Lurie, M. H.: The Neural Mechanism of Hearing: Pathology of the Organ of Corti, Laryngoscope **47**:418-420, 1937.

Corti. He also notes that no degeneration of internal hair cells has been observed in association with normal external hair cells in the same region. Degeneration of external hair cells can be present without that of internal hair cells. Davis, Dworkin, Lurie and Katzman [19] make a preliminary report on the correlation of the behavioral, electric and anatomic changes in the ears of animals with operative lesions in the cochlea. Part of the summary follows.

> Thickening of the round window produced little or no depression of actual hearing but considerable loss of electrical response, probably caused by interference with the flow of electric current. The degree and extent of damage to the organ of Corti revealed by absence of nuclei of the hair cells corresponded to general or local depressions in the electric audiogram. The range of frequencies and degrees of loss of sensitivity were estimated with considerable accuracy in all cases on the basis of the anatomic lesions. The correspondence between results of behavioral tests and the anatomic and electrical changes was good, but not perfect.

Davis and his co-workers also report that in a number of animals with complete destruction of the cochlea and total deafness demonstrable by the behavioral and electrical tests the saccules were normal but there was no auditory response. The saccule does not contribute to hearing or to the electrical tests. This is contrary to the conclusions of Ashcroft and Hallpike that the saccule is an organ of vibration (hearing) sense. Ashcroft and Hallpike state that the results support the suggestion of Professor Tait that the saccule in man is concerned with bone conduction.

Hughson and his co-workers,[20] in a report on the neural mechanism of hearing, conclude that degeneration of the spiral ganglion following section of the auditory nerve does not affect the cochlear response. Ablation of the dorsal and ventral cochlear nuclei, with resulting peripheral and central neural degeneration, had no effect on the cochlear response. Under these conditions the cochlear response was normal, though no action currents were obtained. In the cat the intact organ of Corti can initiate its individual response, regardless of the nerve or ganglia.

The conclusions of Hughson are contradicted by Hallpike, Ashcroft and their co-workers,[21] who observed degeneration of the nerve and

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19. Davis, H.; Dworkin, S.; Lurie, M. H., and Katzman, J.: Behavioral, Electrical and Anatomical Studies of Abnormal Ears, Laryngoscope **47**:435-447, 1937.

20. Hughson, W.; Thompson, E., and Witting, E. G.: The Neural Mechanism of Hearing: Animal Investigations; an Experimental Study of the Neural Mechanism of Hearing, Laryngoscope **47**:480-491, 1937.

21. Hallpike, C. S.; Hartridge, H., and Rawdon-Smith, A. F.: On the Electrical Responses of the Cochlea and the Auditory Tract of the Cat to a Phase Reversal Produced in a Continuous Musical Tone, Proc. Roy. Soc., London, s.B **122**:175-185, 1937. Ashcroft, Hallpike and Rawdon-Smith.[17]

organ of Corti in a cat that gave a normal cochlear response after operation. In a group of animals with marked neural degeneration the response was absent. In a third group there was widespread serous labyrinthitis with much exudate in all the perilymph spaces. In this group the cochlear response was enhanced. From these observations Ashcroft and his co-workers concluded that the cochlear response is the result of polarization of the membranes, and not of activity of the organ of Corti.

Eyster, Bast and Krasno[16] conclude (1) that the cochlear response does not depend on the structural integrity of the organ of Corti or associated structures and (2) that the mechanism is resistant to polarizing current or toxic materials introduced into the cochlea or applied to the round window. They claim that the cochlear response may be simulated by a model in which potentials arise as the result of flow of water through membrane pores, the flow being caused by sound waves sent to the model. They present the view that the flow of endolymph or perilymph through the membranes produces the response. It is difficult to correlate their findings with those of other workers.

The source of the cochlear effect is discussed at length on account of its importance in the interpretation of experiments with the method of Wever and Bray. At present the evidence is in favor of the organ of Corti as the source, though further experimentation may be necessary before the origin of the phenomenon is settled.

The effect of chemicals applied to the round window has been reported on by Fowler and Forbes[22] and by Wever and Bray.[23] They describe definite changes in the organ of Corti, which were most severe at the round window and extended up to the helicotrema, the degree of involvement depending on the strength of the solution used and the time of application. The two reports agree as to the changes observed. Eyster, Bast and Krasno[16] have now come to the conclusion that the effect is one involving the mechanism for middle ear conduction, and not one of direct action of the organ of Corti. These experiments may point to a method for relieving severe tinnitus by introduction of drugs into the cavity of the middle ear.

Attempts to stimulate the human organ of Corti directly by electric means have been reported by the Russian physiologists Andreef, Gersuni and Volokhov.[24] These experiments are of extreme importance to the

22. Fowler, E. P., Jr., and Forbes, T. W.: End-Organ Deafness in Dogs Due to the Application of Certain Chemicals to the Round Window Membrane, Ann. Otol., Rhin. & Laryng. **45**:859-864, 1936.

23. Wever, E. G., and Bray, C. W.: Effects of Chemical Substances upon Electrical Response of the Cochlea, Ann. Otol., Rhin. & Laryng. **46**:291, 1937.

24. Andreev, A. M.; Volokhov, A. A., and Gersuni, G. V.: Electric Excitability of the Organ of Hearing: Effect of Alternating Currents on Patients with Lesions of the Internal or Middle Ear, Fiziol. zhur. **18**:250-264, 1935.

otologist, as they show that it is impossible to restore or improve hearing by an electric apparatus for direct stimulation of the cochlea or the auditory nerve. With stimulation of an injured auditory organ by means of an electrode in the cavity of the middle ear, the following observations on hearing were made: With frequencies of from 100 or 120 to 6000 double vibrations, a smooth buzzing noise occurred, and its character remained unchanged. When the strength of current was increased, it was possible in some cases to observe tonal sensations in addition to sensations of noise. The pitch of the tone was constant during all frequencies.

Brogden and Culler [25] report on the effect of roentgen therapy on the pituitary body. They believe that the increased hearing effect is due to lowering of the sugar content of the body. They also state that diabetic subjects and normal dogs showed increase in acuity of hearing when the blood sugar was lowered and loss when it was raised. These experiments again raise the question of the cause of nerve deafness. Is the pituitary gland the basis?

Brogden, Culler and their co-workers [26] report on the localization of sound in the acoustic tracts up to the medial geniculate bodies in the guinea pig. They conclude that destruction of a single geniculate body is equivalent to destruction of the hemicortex or the cochlea on one side. Incomplete destruction of a single geniculate body causes restricted loss. Localized bilateral lesions affect some frequencies more than others, proving that the several frequencies traverse the geniculate body by different pathways. They conclude from these experiments that the topographic distribution of frequencies which begin in the cochlea persists at least as far as the geniculate body.

Kemp, Coppee and Robinson [27] report the results of their experiments with unilateral and bilateral stimulation of the auditory pathways in the brain stem of cats. The neural thresholds (action currents) appear to be reached at intensities which give a just measurable cochlear response for low tones when the cochlear electrode is on the apex or for high tones when it is on the round window. They made the following observations: With an electrode in the auditory pathways the threshold for neural activity was at the level of that for human audi-

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25. Brogden, W. J., and Culler, E.: Increased Acoustic Sensitivity in Dogs Following Roentgen-Radiation of the Hypophysis, Am. J. Physiol. **119**:13-23, 1937.

26. Brogden, W. J.; Girden, E.; Mettler, F. A., and Culler, E.: Acoustic Value of the Several Components of the Auditory System in Cats, Am. J. Physiol. **116**:252-261, 1936. Culler, E. A.: Topography of the Acoustic System in Cochlea and Medial Geniculate Bodies, Laryngoscope **47**:448-452, 1937.

27. Kemp, E. H.; Coppe, G. E., and Robinson, E. H.: Electric Responses of the Brain Stem to Unilateral Auditory Stimulation, Am. J. Physiol. **120**:304-315, 1937. Kemp, E. H., and Robinson, E. H.: Electric Responses of the Brain Stem to Bilateral Auditory Stimulation, ibid. **120**:316-322, 1937.

bility. Each cochlea is represented bilaterally in the lateral lemniscus. No significant difference was found in thresholds, latencies and maximum amplitudes when homolateral or contralateral placements of electrodes were made. If one ear was stimulated slightly before the other, both responses appeared in the lateral lemniscus with the same difference in time, and the second response showed no loss of amplitude. This observation is of interest with regard to localization of sound; that is, localization depends on the difference in time of stimulation.

Kobrak [28] reports on experiments in which he showed that changes in pressure in the middle ear have a marked effect on the labyrinthine pressure. With use of the tensor tympani muscle response to sound, Kobrak showed that occlusion of the round window causes loss of cochlear stimulation. He concluded that elicitation of the middle ear muscle reflex with acute stimuli furnishes a means of studying hearing. His experiments seem to show that the organ of Corti is responsive to sound reaching it either by the round or the oval window, that the cochlea can be stimulated directly by the air of the middle ear and that the ossicles are not the only pathway by which sound may reach the cochlea but that direct transformation of sound waves of the air in the middle ear into fluid waves in the inner ear is possible.

Wiggers,[29] in a series of experiments with the effect of contraction of the tensor tympani and stapedius muscles on the transmission of sound, reports that the stapedius muscle responds before the tensor tympani muscle and that the contraction of the tensor tympani muscle is overpowering and outlasts that of the stapedius muscle. The muscles can act antagonistically and synergically. Wiggers believes that the muscles may "tune up" the transmission apparatus so that it becomes supersensitive to a few tones of medium frequency, with the optimum at 1500 cycles. The chief function of the muscles of the middle ear is to produce loss of sensitivity to low tones, especially those of great amplitude, and thus to act as a protective mechanism. Wiggers notes no effect of this mechanism on sound above 2000 cycles per second.

Bekesy,[30] in a study of the physical phenomena of the middle ear, ossicles and drum, comes to the same conclusion as that of Kobrak and Wiggers. The middle ear provides a protective mechanism for the inner ear. The unstable equilibrium of the ossicular chain when subjected to large sound pressures changes its pattern of vibration so that movements of the drum are no longer carried over into the fluid of the inner ear.

28. Kobrak, Heinrich G.: Animal Experiments on the Mechanism of the Acoustic Irritation in the Cochlea, Laryngoscope **47**:453-460, 1937.

29. Wiggers, H. C.: The Effect of Contraction of the Intra-Aural Muscles on Transmission of Sound in the Middle Ear, Am. J. Physiol **119**:420, 1937.

30. Bekesy, G. V.: Zur Physik des Mittelohres und über das Hören bei fehlerhaftem Trommelfell, Akust. Ztschr. **1**:13-23, 1936.

This type of vibration when present causes the tickle and pain sensation to appear. Bekesy also states that good hearing for low tones in the absence of the ear drum can be explained by the fact that the sound pressures act both on the stapes and on the round window.

Steinberg,[31] of the Bell Telephone Laboratories, studied the relation between tone frequency and position of stimulation on the basilar membrane, the calculations being based on differential sensitivity to pitch. The positions calculated confirm the placement of pitch perception as obtained from the cochlear response in the guinea pig and from studies on man. Steinberg also reports on the dependence of impairment in hearing on intensity of sound. The method used was measurement of hearing loss for levels of sound well above the threshold for deafness, and therefore audible to the deafened person. The tones were balanced by the observer. In persons with deafened ears who heard loud sound with normal loudness the deafness could be represented quantitatively on the assumption that it was due to atrophy of the nerve. In the presence of noises the loudness judgment for normal ears and that for nerve-deafened ears were found to be similar. These findings are of great interest, as they seem to confirm the observations by Lurie on the difference in response between internal and external hair cells. Fowler [32] has presented the clinical aspect of this work in its relationship to fixation of the stapes.

Davis and Stevens [33] measured the combination tones in the electrical activity of the cochlea. The tones used were 700 and 1,200 cycles per second, and the animals were stimulated by the two tones at the same time. The resulting electrical activity of the cochlea was analyzed by means of an electric wave analyzer. The authors found many combination tones present in the cochlea. The sum and difference tones, of 500 and 1,900 cycles, were found to grow with the fundamental tones. The tones of 1,700, 200, 3,100 and 2,600 cycles followed the growth of the harmonic tones. The other combination tones were too small for accurate determination. It is of interest to note that differences, summations and harmonic differences and summations can be detected by the ear, even though they are not present in the physical sound heard. This work also gives support to the theory that the cochlea is an analyzer of sound.

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31. Steinberg, J. C.: Positions of Stimulation in the Cochlea by Pure Tones, J. Acoustic Soc. America **8**:176-180, 1937. Steinberg, J. C., and Gardner, M. B.: The Dependence of Hearing Impairment on Sound Intensity, ibid. **9**:11-23, 1937.

32. Fowler, E. P., Sr.: The Diagnosis of Diseases of the Neural Mechanism of Hearing by the Aid of Sounds Well Above Threshold, Laryngoscope **47**:198-300, 1937.

33. Davis, H., and Stevens, S. S.: The Measurement of Combination Tones in the Electrical Activity of the Cochlea, Am. J. Physiol. **119**:296, 1937.

Guild [34] summarizes the present lack of knowledge in regard to the physiologic and anatomic relations of the inner ear. It is not known how sound waves cause stimulation of the nerve. The physiologist has not considered the possibilities presented by movements within the organ of Corti during passage of sound waves through the fluids of the cochlea. The biochemical reactions of the inner ear are almost completely unknown.

Lewy and Kobrák [35] produced small lesions in the cochlear spirals of rabbits, and the ensuing nerve degenerations were traced by the Marchi method. Anatomic differences between the fiber projections of the basilar part of the cochlea, especially near the fenestra rotunda, and those from the rest of the cochlea suggest a structural basis for physiologic objections to the frequency theory.

Mink,[36] on the basis of an extensive survey of the literature and the results of his observations, concludes that all hearing involves pressure on the drum, the pressure being due to sound. The cochlea is a measuring apparatus for the intensity of sound. Of especial importance for transmission of pressure are the ossicles. The cochlea measures the intensity of sound, the sensitivity of the cochlea to pressure being greater than that of the retina to light.

Guttich [37] found that determination of the direction of sound is of great importance in the animal world. Even in lower animals, such as the crayfish, reaction to sound direction is present. Guttich's contentions agree with those of Tulio. He concludes that in the determination of sound direction there exists close teamwork between the cochlear and the neck and vestibular reflexes acting on the eye.

Wittmaack,[38] in a discussion of the structure and function of the tectorial membrane, states that the organ of Corti and the tectorial membrane form a closely connected tissue complex, which is shut off from the endolymphatic space. Antemortem and postmortem experiments indicate that in life there is no separation between the tectorial membrane and the organ of Corti but that they separate shortly after death. The periodic changes in pressure caused by the periodic vibration of the foot plate of the stapes bring about corresponding changes in

34. Guild, Stacy: Comments on the Physiology of Hearing and the Anatomy of the Inner Ear, Laryngoscope **47**:365-372, 1937.

35. Lewy, F. H., and Kobrak, H.: The Neural Projection of the Cochlear Spirals on the Primary Acoustic Centers, Arch. Neurol. & Psychiat. **35**:839-852 (April) 1936.

36. Mink, P. J.: Das innere Ohr als Tastorgan, abstracted, Zentralbl. f. Hals-, Nasen- u. Ohrenh. **29**:225, 1937.

37. Guttich, A.: Nachruf auf Obermedizinalrat, abstracted, Zentralbl. f. Hals-, Nasen- u. Ohrenh. **28**:4, 1937.

38. Wittmaack, K.: Ueber Bau und Funktion der Membrana tectoria, Acta oto-laryng. **24**:397-423, 1936.

pressure in the fluid of the inner ear, which cause the basilar membrane and the membrane of Corti to vibrate with equal frequency, amplitude and phase.

During this process there is no change between the vibration of the organ of Corti and that of the tectorial membrane. From this Wittmaack concludes that freely protruding hairs of sensory cells do not exist. As a fact, the sensory hairs would be superfluous, since all structures embedded in the vibrating fluid must vibrate in the same manner with this fluid. From the foregoing statements it is obvious that the unmyelinated nerve fibers embedded in the fluid space of the organ of Corti could well take up the vibrations of the fluid directly.

This startling observation by Wittmaack is contrary to the modern point of view of the function and interrelationship of the organ of Corti and the tectorial membrane. He has contributed so much to the physiology of the tonus and the mode of stimulation that serious thought must be given to his present surmise. For those who in numerous microscopic sections have seen the hairs protruding into the space between the organ of Corti and the membrane, his conclusions are difficult to accept. With respect to the mode of stimulation of the sensory cells, however, this assumption is logical.

VESTIBULAR PHYSIOLOGIC REACTIONS

Hasegaw [39] states that the static reflexes and the reflexes concerned with rotatory movements can still be elicited in frogs without otoliths. Reactivity to progressive movements is no longer visible in absence of the otoliths. The function of the otoliths in the frog is the same as that in the guinea pig.

In view of the finding by some authors that extirpation of the macula utriculi alone causes disturbances identical with those produced by total extirpation of the otolith organs, Werner [40] raises the question whether the macula utriculi is sufficiently differentiated anatomically to account for multidirectional sensitivity to static changes. A review of his own anatomic researches, as well as those of others, leads him to answer the question in the affirmative. The nature of the stimulus for the sensory epithelium is discussed, and current thories are summarized.

Ferraro, Barrera and Blakeslee [41] found that in monkeys extensive damage of the vestibular nucleus on one side produces vestibular symp-

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39. Hasegaw, T.: Labyrinthreflexe nach Abschleuderung der Otolithenmembranen, Arch. f. d. ges. Physiol. **236**:589-593, 1935.

40. Werner, C. F.: Ueber die statische Funktion der Macula utriculi und ihres Otolithen, Acta oto-laryng. **24**:253-270, 1936.

41. Ferraro, A.; Barrera, S. E., and Blakeslee, G. A.: Vestibular Phenomena of Central Origin: An Experimental Study in Macacus Rhesus, Brain **59**:466-482, 1936.

toms identical in type with those associated with unilateral labyrinthectomy or unilateral section of the eighth nerve, but of greater intensity and longer duration. Similar vestibular phenomena of equal severity result from lesions of the vestibular nuclei on one side after bilateral labyrinthectomy or bilateral section of the eighth nerve. Section of fibers connecting the cerebellar roof nuclei with the vestibular nuclear complex produces typical vestibular signs, which are unmodified by previous bilateral labyrinthectomy. Further, the effects of large unilateral lesions of the vestibular nuclei were unmodified by section of the second, third and fourth posterior cervical roots.

Van Gehuchten [42] believes that the vestibular system is the chief center for the maintenance of equilibrium. He discusses in considerable detail the fiber tracts between the end-organs and the various nuclei distributed in the midbrain and cerebellum, as well as in the spinal cord. He finds an intimate, intricate connection between all the nuclei associated with the vestibular organ.

Cantele and Pais,[43] in experiments on twelve guinea pigs, examined the vestibular reflexes after they had induced passive congestion in the heads by suspending the animals head down for varying lengths of time. The vestibular reflexes first became hypo-active and then disappeared completely. Hypo-activity began usually before the fifteenth hour after the animals were suspended and reached its maximum between the twentieth and the thirtieth hour, when it disappeared completely. When the animals were replaced in normal position after the suspension, the reflexes were again normal. Guinea pigs suspended in this manner for several months, with short periods of rest in normal positions, were still capable of responding in a normal way. Microscopic changes, such as hemorrhages, congestion and edema, were far more marked in the cochlear than in the vestibular portion of the labyrinth.

T'ang and Wu [44] found that in the rat symptoms of cocainization of the nonauditory labyrinth on one side were similar to those of unilateral labyrinthectomy, but were transient. The eye on the side of operation deviated ventrally, and the other, dorsally. There were temporary nystagmus and bending of the head toward the side of operation. When the rat was held head downward, the tail made circular movements. The limbs on the normal side were more extended and abducted than those on the other. The animal fell more easily to the side of operation; it could not right itself promptly when placed on that side.

42. van Gehuchten, P.: Anatomie des voies centrales du système vestibulaire, abstracted, Zentralbl. f. Hals-, Nasen- u. Ohrenh. **29**:162, 1937.

43. Cantele, P. G., and Pais, Carlo: Effetti sperimentali sul labirinto della stasi passiva cefalica (nota preventiva), Arch. ital. di otol. **3**:107-114, 1937.

44. T'ang, Y., and Wu, C. F.: The Effects of Unilateral Labyrinthectomy in the Albino Rat, Chinese J. Physiol. **10**:571-598, 1936.

There were temporary concavity of the body and rolling and circus movements toward the side of operation. The rat always rolled when it was submerged in water. The reaction of the head and eyes to rotation differed with the direction. The tonus of the limbs was not affected by blindfolding or bending the head. The animal could right itself in the air when dropped. Cocainized rats showed head nystagmus.

LeHeux and de Kleyn [45] observed that if a rabbit in a state of acute alcohol poisoning was placed on its side definitely directed spontaneous nystagmus was elicited, the direction being dependent on the position of the head in space. The authors made observations on the isolated external rectus muscles and found strange group responses. Besides, they made the remarkable observation that if nystagmus had disappeared when the rabbit was in the left lateral position it recurred when the animal was laid on its right side; if it had disappeared in this position, it could be reelicited by turning the rabbit back on the left side. The authors consider this phenomenon to be analogous to the inductive phenomena of Sherrington associated with spinal reflexes and liken it to the rhythmic vestibular reflexes of Wodak.

Ter Braak [46] measured angular accelerations which were necessary to elicit eye movements in rabbits. An unusual feature of these experiments, it may be mentioned, was a small mirror fastened to the cornea of the experimental animal, by which the deviations of an incident beam of light were used for measurement of the ocular movements. If the angular acceleration is slight, the rabbit reacts with nystagmus or deviation of the eyes. Acceleration of less than 0.25 degree per second per second was sufficient to elicit deviation of the eyes. In an exceptional case, 0.09 degree per second per second was sufficient to elicit a reaction. The latent period between the beginning of acceleration and that of the ocular reaction is, even in slight acceleration, much less than would be expected on the basis of the results of other workers.

In another article ter Braak [47] discusses the question of what physical conditions must prevail if the vestibular apparatus can be stimulated by rectilinear operation. He concludes that rectilinear acceleration can cause deviation of the cupola if the specific gravity of the latter differs from that of the endolymph. If the difference in specific gravity is greater than 1 part in 10,000, a rectilinear acceleration of 981 cm. per second (acceleration by gravity) would be sufficient for stimulation.

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45. LeHeux, J., and de Kleyn, A.: Untersuchungen an den isolierten Augenmuskeln des Kaninchens während des Lagenystagmus bei akuter Alkoholvergiftung, abstracted, Zentralbl. f. Hals-, Nasen- u. Ohrenh. **29**:228, 1937.

46. ter Braak, J. W. G.: Ueber die Empfindlichkeit des Bogengangsapparates für Winkelbeschleungigungen, Arch. f. d. ges. Physiol. **238**:319-326, 1936.

47. ter Braak, J. W. G.: Kann der Bogengangsapparat durch geradlinige Beschleunigung gereizt werden? Arch. f. d. ges. Physiol. **238**:327-332, 1936.

At the Fifth Annual Meeting of the Hungarian Oto-Laryngological Society, there were several contributions to the physiology of the vestibular apparatus. A paper by Germán,[48] though not available for abstracting, is apparently worth looking into.

Blohmke [49] reported that it has been shown in dogs placed in the dark at birth that so-called dark or twilight nystagmus, once it has arisen by optic channels throughout the cortex, continues even after the entire cerebrum is removed. A subcortical mechanism is therefore the basis of this form of nystagmus. This discovery led to further experiments concerning the genesis of vestibular nystagmus. Though the slow component of nystagmus is generally looked on as a purely vestibular reaction to stimulus, opinions still vary as to the nature of the quick component. Consequently, local electric stimuli were applied to the caudex cerebri of the rabbit after removal of the whole cortex. In this way regularly appearing nystagmoid movements showing the same character as vestibular nystagmus were produced. Further, by means of section of the thalamus and the anterior corpora quadrigemina, regularly appearing, spontaneous nystagmus was produced. It was assumed accordingly that frontal to the eye muscle corpuscles there is a system of neurons in the midbrain (thalamus and anterior corpora quadrigemina) which exercises a predominating influence on the activity of the apparatus governing ocular movement, in giving rise to the nystagmic movements of the eyes. Examination of the rabbit brain led to the belief that the columns of the formatio reticularis in the caudex cerebri and pons cerebelli were the site of origin, and the nystagmus was traced to a disturbance in the coordination of this short fiber system and the long channeled system of the fasciculus longitudinalis inferior. Later anatomic investigation of the role of the fasciculus longitudinalis inferior and its relations to the nuclear region of the vestibule and the caudate nucleus (striatum), as well as the role of the fasciculus retroflexus and its relations to the important nerve endings of the nervus opticus in the roof of the anterior corpora quadrigemina, has, together with Musken's comprehensive exposition of the supravestibular system, thrown fresh light on the central mechanism governing the process of the two aforementioned ocular movements, i. e., twilight and vestibular nystagmus.

Fleischmann [50] reported also on a series of experiments with parallel calorization of the same subject to ascertain whether changes in the temperature, the amount of fluid injected and the length of time during

48. Germán, T.: The Present Position of Research on the Vestibular Function, Orvosképzés, 1937, supp.

49. Blohmke, A.: On the Subcortical Source of Vestibular Nystagmus and of Twilight Nystagmus, Acta oto-laryng. **25**:489, 1937.

50. Fleischmann, L.: Comparative Investigations into Caloric Sensitivity, Acta oto-laryng. **25**:490, 1937.

which the ear is syringed influence the extent of the reaction. It was found in all three series of investigations that, despite alteration of the stimulus, there was in the majority of cases no significant change in the reaction and that only in the minority did the weaker stimulus provoke a weaker reaction. This striking result is probably to be explained by the slight difference in the stimuli, and it indicates a need for caution in application of the caloric method to qualitative labyrinth tests.

There were also papers by Barkó, Alföldy, Kerekes and Kelemen, with a discussion.

VASOMOTOR PHENOMENA

Frazer [51] shows that the internal ear can be completely deprived of vasoconstrictor impulses by section of the truncus sympatheticus in the neck. Vertigo and tinnitus are the two conditions for which this operation is at times indicated. The theoretical basis for certain forms of vertigo and tinnitus is a vasospastic condition in the internal ear, the symptoms being produced by the sudden influx of blood into the labyrinth as the result of sudden vasodilatation following spasm of the vessels. The sympathetic system has been shown to control the tone of the labyrinthine vessels. Various operations, such as decortication of the common carotid arteries, excision of the cervical portion of the sympathetic trunk and the ganglia and combinations of these procedures, have been tried, with temporary success. Perhaps this is true because no one before Frazer has worked out the exact course pursued by the sympathetic fibers in reaching the internal ear.

Frazer, in a series of experiments with cats, stimulated various parts of the sympathetic chain in the neck. During stimulation he injected intravenously a 4 per cent solution of bromophenol blue and promptly killed the animal. The body tissues were stained an intense blue except where stimulation of vasoconstrictor fibers had caused local ischemia. There the tissues were white with a definite line of demarcation separating them from the unstimulated areas.

Stimulation of the stellate (first thoracic) ganglion, the cervical portion of the sympathetic trunk or the superior cervical sympathetic ganglion gave identical results, i. e., vasoconstriction of the external, middle and internal ears. These results were unaffected by decortication of the internal and external carotid arteries. If the cervical portion of the sympathetic trunk is cut, however, stimulation, below this level does not produce vasoconstriction anywhere in the ear. This excludes the possibility that vasoconstrictor fibers reach the ear from the stellate ganglion via the periarterial plexus of the vertebral artery.

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51. Frazer, Philip: The Vasomotor Control of the Ear, J. Laryng. & Otol. **51**: 579-585, 1936.

The practical importance of Frazer's work is the demonstration that no vasconstrictor fibers reach the ear via the periaterial plexuses of the carotid or the vertebral artery. All vasoconstrictor impulses to the ear travel in the cervicosympathetic trunk. Therefore operation to deprive the ear completely of vasoconstrictor stimulation need consist only of division of the truncus sympatheticus. To prevent regeneration of fibers, however, the operator should remove a section of the truncus.

Kotyza [52] discusses the vestibular apparatus and the vegetative nervous system but has a more pronounced influence on the parasympathe following conclusion: 1. The vestibular apparatus exercises a considerable influence on the vegetative nervous system.

2. In most cases rotation increases the tonus of the vegetative nervous system, but has a more pronounced influence on the parasympathetic than on the orthosympathetic system.

3. Excitation called forth by the Kobrak caloric test generally decreases the tonus of the vegetative nervous system, but acts in a more pronounced way on the orthosympathetic than on the parasympathetic system.

4. Observations confirm the theory of Lagnel-Lavastin concerning the functional relationship of the orthosympathetic and the parasympathetic nervous system.

Stimulation of the labyrinth by means of rotation is more marked than that by the caloric test. Sudden stimulation of the vestibular nervous system increases the tonus of the vegetative nervous system and, above all, the so-called parasympathetic system.

The least stimulation of the vestibular nervous system, according to Kobrak, decreases the tonus of the vegetative nervous system. The more pronounced susceptibility of the orthosympathetic system may be explained by the fact that warm water syringed against the upper frontal wall of the acoustic meatus in the Kobrak test stimulates the vagus nerve, through intermediation of the auditory branch.

CLINICAL APPLICATIONS OF VESTIBULAR PHYSIOLOGIC REACTIONS

Tumarkin [53] says that the main sources of error arising from the rotation test are: (1) excessive stimulation; (2) visual influences; (3) personal bias of the examiner, and (4) confusion of postrotatory nystagmus with: (*a*) per-rotatory nystagmus and (*b*) secondary nystagmus. From his own experiments he finds that the probable threshold of acceleration is about 0.2 degree per second per second. Buys gave a value

52. Kotyza, F.: L'appareil vestibulaire et le système nerveux végétatif, Acta oto-laryng. **25**:51-60, 1937.

53. Tumarkin, J. A.: Some Observations on the Function of the Labyrinth, Proc. Roy. Soc. Med. (Sect. Otol.) **30**:599-610, 1937.

of from 1 to 2 degrees per second per second, and others, as much as 8 degrees per second per second. Tumarkin stresses the point that the specific stimulation for the semicircular canals is acceleration, and not velocity. He does not approve of the use of Bartel's glasses to overcome visual influences on the nystagmus and prefers his own modification of the nystagmograph, designed by Professor Dodge, of Yale University. Its value lies in the fact that the eyes are closed, eliminating all visual influences, and that the per-rotatory nystagmus (nystagmus elicited in the beginning of the acceleration) can be measured. Tumarkin objects to the sudden stopping of rotation as too violent a stimulus; it is his aim to bring the rotation test to greater refinement.

The author describes two types of Ménière's syndrome.

	Syndrome of Semicircular Canals	Syndrome of Otolith Apparatus
Onset	Ingravescent	Instant
Duration	Hours to days	Minutes or less
Nausea, pallor, etc.	Present	None
Disorientation	Present	None

The two types exist, but it is doubtful whether Tumarkin proves in his discussion where the source of the trouble is, particularly in view of what Dandy and Crowe have said.

McNally [54] for a number of years has utilized the tilt table for testing the function of the semicircular canals. Reference is made to numerous reports on his ablation experiments with frogs; he has come to believe that this test is of great importance and should be used more frequently in clinical work. He has shown that "a slow tilt stimulates the utricle but does not stimulate the semicircular canals, and in response to a slow tilt the utricles show a compensatory reaction. A quick tilt stimulates the vertical semicircular canals to bring about a prompt compensatory reaction of the body musculature."

An important phase of his discussion is the description of the so-called modes of utricular stimulation, namely, the first and the second mode of stimulation. The first is in response to a slow tilt, in which there is a compensatory reaction of the body, head and eyes; the second is in response to a quick tilt, in which there is a prompt movement of the head and body in the direction of the tilt—an anticompensatory reaction.

There are a brief description of the mechanism of semicircular stimulation and a fuller discussion of the mechanism of utricular stimulation. McNally also describes the reaction pattern of the body musculature. He does not take the saccule into consideration in analyzing the reaction which follows vestibular stimulation, because it has not been established to have any connection with the equilibrial mechanism.

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54. McNally, W. J.: Labyrinthine Reactions and Their Relation to Clinical Tests, Proc. Roy. Soc. Med. (Sect. Otol.) **30**:905-916, 1937.

In his abstract he states:

> If, in addition to the usual equilibrial tests, the quick tilt test is used in this way and a careful analysis is made of the reactions of patients with labyrinthine or intracranial lesions, diagnosis of lesions of individual labyrinthine end-organs or of their intracranial connections may become a routine procedure in the clinic just as it is now possible in the laboratory.

Mowrer [55] experimented with human subjects to learn whether vision during rotation has any influence on postrotational nystagmus. The following methods were employed:

1. The subject was rotated in the dark and then in the light. Postrotational nystagmus was observed in the light in all cases.

2. The subject was rotated in a small, internally illuminated visual environment, which moved with the subject. This eliminated light adaptation after the test.

3. The subject looked at his finger tip, which rotated with him.

4. The subject looked through a prismatic pseudoscope, which could be adjusted to reverse the direction of the object or to make it move twice as fast in the same direction, or even to change the plane of motion to the vertical.

It was observed that the only change occurred when the subject was in darkness during rotation, postrotational nystagmus being much less when vision was permitted.

Mowrer discusses the mechanism of vestibular nystagmus after stimulation as demonstrated by various investigators; he assumes that both the vestibular receptors and the vestibular nuclei are capable of afterdischarge, by independent mechanisms.

Cinelli [56] gives a practical review of the clinical application of the labyrinthine tests. He bases the tests on the physiologic reaction established by Flourens and Ewald. The clinical application of the tests is based on the work of Bárány.

The article gives the classic description which has been handed down from one textbook to another, and one must admit that it is the only workable method for the great majority of clinicians; if it serves its purpose in that respect there can be no dissension. On the other hand, it does not discuss the refined methods of testing the labyrinth; results, therefore, may be misconstrued.

There are excellent diagrammatic sketches of the intracranial connections of the eighth nerve and the ocular, as well as the vestibular,

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55. Mowrer, O. H.: The Influence of Vision During Bodily Rotation upon the Duration of Post-Rotational Vestibular Nystagmus, Acta oto-laryng. **25**:351-363, 1937.

56. Cinelli, A. A.: Labyrinth Reactions and Their Clinical Importance, Laryngoscope **46**:815-839, 1936.

nuclei. The summary and differentiation between involvement of the central and that of the peripheral nerves are good.

If the author would forewarn his readers that the labyrinthine tests are not conclusive as they are practiced today but that the results, in association with other signs and symptoms, may give a lead in making a diagnosis, he would do much to take away the certainty of their infallibility that seems to exist.

I commend this article to the average reader, who does not inquire too deeply into the physiologic response of the reactions.

Lorente de Nó [57] states that Bárány's theory of caloric stimulation of the endolymph is understood and accepted but that there is much to be done before its practical application can be brought into accord with simple and invariable rules.

Caloric stimulation creates different strengths of stimulation in the three canals. The horizontal canal is stimulated most strongly; the anterior, next, and the posterior, least. This is due to distance from the point of stimulation.

A. *Measurement of the Strength of the Nystagmic Reaction.*—The speed of the slow component varies with the strength of the stimulus. The number of strokes and the duration of the nystagmus indicate the intensity of the stimulus. Each subject reacts with wide variation to a given stimulus. Nystagmus itself is a rhythmic reflex response of the nervous system to labyrinthine stimulation.

This brief summary of known facts leads to an important conclusion, namely, that measurements of the strength of nystagmus, expressed by its duration and number of strokes, made on different subjects, and even on the same subject under different conditions, cannot be considered sufficiently reliable to establish a means of comparison and to determine whether the labyrinth is in a normal or a pathologic condition. It is evident that in extreme cases valuable information can be secured, but in general these measurements have limited diagnostic value.

With the caloric test the situation is still worse, because the individual variations are as great as those with the rotatory test, or even greater. This is due perhaps to the fact that stimulation of the labyrinth in this test differs from that under physiologic conditions in three important points. First, the stimulus (endolymph current) lasts for a long time—many times longer than ever occurs in free life. Second, only one labyrinth is stimulated, while under physiologic conditions the nervous system receives simultaneouly impulses from both labyrinths. Third, the test is made with positions of the head which cause an

57. Lorente de Nó, R.: Theoretical Basis and Technic of the Caloric Test, Tr. Am. Laryng., Rhin. & Otol. Soc. **42**:377-393, 1936.

improper balance of the stimuli arising from the semicircular canals and from the otolith organs.

B. *Latency of the Caloric Nystagmus.*—The strength of a reflex can be measured in terms of its latency. The latency of caloric nystagmus consists of three intervals: (1) the time necessary for transmission of heat to the labyrinth; (2) the time necessary for the endolymph current to attain threshold speed, and (3) the true latent period of the vestibular action.

Measurements of the true latency of caloric nystagmus are in general valueless because one is not sure of the exact time that the stimulus is applied.

C. *Duration of Caloric Nystagmus.*—This depends on the duration of the flow of endolymph and not on the intensity of the stimulation, i. e., cold or heat. Changes in the central nervous system may create prolonged nystagmus of from two to three times the usual duration which is of value clinically. Nystagmus of short duration is of no diagnostic value.

D. *Practical Use of Caloric Nystagmus.*—The caloric test is of great importance for determination of the state of balance of the two labyrinths, as normally they act antagonistically and are accurately balanced.

Vogel's method of stimulating the labyrinth is simple and accurate. Its theoretical basis is as follows:

1. Normally, equal stimulation of the right or of the left labyrinth will give rise to equally strong nystagmus, in opposite directions.

2. Irrigation of each labyrinth with cold and warm water will cause nystagmus in opposite directions.

3. By comparing the nystagmus obtained after irrigation of each labyrinth with both cold and warm water, one can determine whether the two labyrinths are equally excitable and whether the lack of balance is accompanied by involvement of the vestibular centers.

An irritative lesion of one labyrinth will give accentuated nystagmus to that side, while a destructive lesion that has not destroyed completely the excitability of the labyrinth will give accentuated nystagmus to the opposite side. Each shows latent nystagmus to the side accentuated.

If the irritability of one labyrinth is diminished, as when the eighth nerve is gone, there is no response in the ear on the same side but there is accentuation of nystagmus to the side of the other ear when it is stimulated.

E. *Technic of the Caloric Test.*—The head is placed in the optimal position (with the external semicircular canal vertical in space), and the external auditory canal is irrigated for five, ten or fifteen seconds with water at 10 or 20 C. (18 or 36 F.) above or below body temperature.

A 0.3 mm. stream is directed toward the posterosuperior wall of the canal. Only the time that the water is flowing, not the quantity of water, is recorded. The eyes are observed through Bartel's spectacles. The number of strokes of nystagmus are counted, and the duration of the nystagmus, starting and ending with the movements of the eyes, is measured by a stop watch. The strength of the nystagmus is then measured in terms of two values—the number of strokes and the duration, expressed in seconds. Irrigation for five seconds with water at 10 C. above or below body temperature produces weak nystagmus. Irrigation for ten seconds is best. If irrigation for fifteen seconds with water at 20 C. below body temperature produces no nystagmus, it is rare that stronger stimuli will do so.

Abstracts from Current Literature

Ear

ELEMENTS OF NERVES AND GANGLIA OF THE INTERNAL EAR. I. ERNYEI, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **141**:343, 1936.

Ernyei investigated two problems: 1. Does the internal ear contain sympathetic fibers, and if so, what quantitative relationship exists between the medullated and the nonmedullated fibers? 2. What are the characteristics of the cells of the vestibular and cochlear ganglions? The studies were made on material obtained from young guinea-pigs, cats and dogs. The fresh materials of the internal ears were fixed, decalcified, embedded in pyroxylin and paraffin and sectioned. The longitudinal and transverse sections were treated either by the prolonged osmium method or with a silver preparation. The longitudinal as well as the transverse sections of the vestibular nerve revealed medullated, thin-sheathed fibers. In the vestibular ganglion there are two types of ganglion cells: large light ones and small dark ones. In the cochlear nerve there are fibers with thin medullar sheaths. Near the vessels there are nerves consisting only of nonmedullated fibers. The cochlear ganglion contains two types of ganglion cells: lighter ones and darker ones. It appears probable that in the mechanism of the vasomotor labyrinthine reflexes, the nonmedullated fibers of the sympathetic nervous system play an essential part.

EDITOR'S ABSTRACT.

ANATOMIC PATHS IN ORBITAL COMPLICATIONS OF OTOGENIC SINUS THROMBOSIS. W. G. BLÅNKSTEIN, Monatschr. f. Ohrenh. **70**:798 (July) 1936.

Blankstein reports a case in which a metastatic abscess developed in the left eyelid as a result of otogenic thrombosis of the transverse sinus of the same side. In answer to the question as to which path the infection followed, he says that it could not have been spread by way of the lymph channels, since in that case there would have existed a collateral edema of the face and also the manifestations of lymphangitis. A retrograde infection by way of the sinus cavernosus likewise seems improbable to the author, for thrombosis of the corresponding sinuses would have developed. He is inclined to believe that the infectious material was transported by the venae diploicae, pointing out that the anastomoses between the venae diploicae and the veins of the eyelids would be the easiest way by which the particles of the thrombus could be transported.

EDITOR'S ABSTRACT.

ROENTGEN PICTURE AND SURGICAL ASPECTS OF THE MASTOID IN ACUTE OTITIS MEDIA. L. BENSCH, Ztschr. f. Hals-, Nasen- u. Ohrenh. **39**:502, 1936.

Bensch points out that opinions about the value of roentgenoscopic examination in cases of acute otitis media differ greatly. Whereas some reject this form of examination entirely, others consider it the decisive factor in determining the therapeutic procedure. The author studied the reliability of the roentgen picture and the causes of erroneous roentgen diagnosis in 209 surgical cases at the oto-rhinolaryngologic clinic of the University of Leipzig. He shows that erroneous roentgen diagnoses can be caused, for instance, by the presence of the pathologic process in a region not readily accessible to roentgen rays, as in the region of the antrum or of the posterior wall of the auditory meatus, in the retrolabyrinthine portions, in the deeper parts of the mastoid or in the retrosinal region. An anatomic abnormality, such as an abnormal position of the sinus or a fissura mastoideosquamosa, may likewise lead to an erroneous roentgen diagnosis. Other factors that may play a part in mistaken roentgen diagnoses are a spongiose

mastoid, irregular pneumatization, smallness of the resorptive bone process, abnormally small anatomic structures, slight contrast shadows of abscesses, and deficiency of contrast in infantile and senile temporal bones. In view of these factors, the author thinks that the roentgen aspects can be utilized as an indicator for surgical treatment only in connection with the clinical aspects. If the clinical symptoms are severe, antrotomy should be performed even though the roentgen picture may not reveal anything significant. EDITOR'S ABSTRACT.

THE POINTING TEST. WILHELM BEHRMAN, Upsala läkaref. förh. **42**:67 (July) 1936.

This extensive investigation deals with the clinical value of the pointing test. Behrman believes that contradictory reports and opinions are due to the fact that no standard methods of testing are used. In addition, there exists no accord among clinicians regarding the value of the pointing test as a clinical method. The difficulty lies also in the fact that no attempt has been made to ascertain the range of average deviation in the performance of the tests in normal persons. Behrman states that since there is no way of telling how far a person will past point without stimulation, it is hard to determine whether the same person will point differently with stimulation.

In order to secure some notion concerning the range of normal variability, Behrman made use of statistical methods. He established first the average effect of a given type of stimulation in a number of normal persons and then the constancy of the effect of a given stimulation in an individual case. His investigation of spontaneous pointing in normal persons leads him to believe that the results are determined by the methods used. He tried eleven modifications of the original technic of Bárány. One is a corrected pointing test, with the subject's head bent backward and one arm moving downward toward a table, the top of which is replaced by a plate of glass. Another method is uncorrected upward pointing toward the plate, and the third method, uncorrected upward pointing with both arms toward the plate. The glass was fitted into a table top for visibility and recording. Behrman found that in upward movement toward the goal there was drifting of the arms from the midline outward. In the third method there was an average deviation of 3.79 ± 0.08 (0.25) cm. for the right arm and 3.33 ± 0.08 (0.25) cm. for the left. With other methods in which the goal is reached by a downward movement, no definite outward deviation or only deviations inward were noted. Behrman therefore comes to the conclusion that the general view according to which the arms always have a tendency to deviate spontaneously outward is erroneous.

He found further that in pointing tests in which correction toward the goal is made there is a mild range of variation of from about 10 to 15 cm., while in uncorrected methods the variation is twice as large. He also observed that methods with the tendency for spontaneous past pointing outward show an increase in past pointing for the right arm and none for the left.

Study of the average effects of stimulation showed that past pointing takes place invariably in the direction established by Bárány and other investigators. After caloric stimulation or rotation past pointing is in the direction of the slow phase of the nystagmus. When the head is turned, deviation is in the opposite direction, and when the head is bent, deviation is in the same direction. The degree of deviation following stimulation depends on the methods used; it is greater when tests are used which require upward movements of the arms, especially when no correction is made after each pointing. This effect is more pronounced when the person tested points with both arms simultaneously. Behrman found further that after calorization deviation of spontaneous pointing from the range of the normal average increases slowly, reaches its maximum at about the tenth pointing and decreases gradually from then on. As an example, he offers the following observation: The effect of stimulation following rinsing the left ear with 100 cc. of water at a temperature of 20 C. was 5.22 ± 0.56 cm. for the right arm at the first pointing, while at the tenth pointing it increased to 11.66 ± 0.81 cm. The

corresponding figures for the left arm were 2.71 ± 0.62 cm. and 7.97 ± 0.79 cm., respectively. After rotation the type of deviation was entirely different. The greatest deviation was at the first pointing and decreased in the subsequent tests. The effect following rotation to the right was 9.93 ± 0.87 cm. for the right arm at the first pointing and 3.82 ± 0.74 cm. at the second trial. The corresponding figures for the left arm were 9.03 ± 0.88 cm. and 4.91 ± 0.74 cm., respectively.

Behrman claims that the method of testing determines which arm will show the greatest reaction to a definite vestibular stimulation. One method may produce the greatest influence on the homolateral arm, and another method, on the contralateral. He found that rinsing the ear with cold water affects chiefly the opposite arm, while rinsing with warm water affects the homolateral arm. Rotation has its greatest effect on the arm which is opposite the direction of rotation. All these observations are formulated in a rule according to which in the test of pointing the strongest reaction following rotation or calorization will be in the arm which must deviate mesially when it deviates in the direction of the slow component of nystagmus. Rinsing with large quantities of water (100 cc.) produces stronger reactions than rinsing with small quantities; stronger reactions are also obtained with cold water. Behrman concludes that the diagnostic value of the pointing test is enhanced through the use of repeated stimulation tests in the same person. He showed further that the range of variation of pointing increases considerably after stimulation. Occasionally this range of variation is twice as large after stimulation as that in spontaneous pointing. Behrman was unable to establish any definite relationship between the onset of dizziness and the degree of past pointing. No relationship could be established between the direction of the apparent movement and that of the deviation.

NOTKIN, Poughkeepsie, N. Y. [ARCH. NEUROL. & PSYCHIAT.]

Larynx

CASEOUS EMPYEMA OF SACCULUS VENTRICULI LARYNGIS IN LARYNGEAL TUBERCULOSIS. F. ZÖLLNER, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **141**:327, 1936.

Zöllner presents a case in which the diagnosis was difficult. The localization of a tumor on the neck in the absence of other symptoms suggested at first a lobus pyramidalis glandulae thyreoideae or a cyst originating on the thyroglossal duct. However, observation over a longer period suggested that the inflammatory process of the laryngeal mucous membrane might be tuberculous. Repeated roentgenographic examination of the lung disclosed a tuberculous process, and when puncture of the swelling yielded caseous pus a causal connection between the pulmonary process and the peculiar tumor seemed to be established. Another important factor in the correct diagnosis was the localization, for the author says that the protrusions of the laryngeal sacculus, which become visible on the outside, either perforate the hypothyroid membrane or push it forward. At any rate, the protrusion of the laryngeal sacculus emerges outward between the hyoid bone and the thyroid cartilage, near the median line. An operation was performed, but only the external portion of the laryngeal sac was removed, radical removal of the pedicle and of the internal portion being impossible on account of extensive adhesions. In discussing the pathogenesis, the author suggests that the protrusion of the laryngeal sacculus resulted from infection of the sacculus with tuberculosis. He thinks that the free opening was probably closed by tuberculous changes and that the resulting stasis of glandular secretion and of disease products resulted in the protrusion of the sacculus. In view of the fact that the tuberculous changes within the closed laryngeal sacculus were more advanced than those of the laryngeal mucous membrane, he assumes that the tuberculosis became established first (probably as the result of a hematogenous process) in Morgagni's ventricle and that in the later course the process became diffused over the mucous membrane at the entrance of the larynx.

EDITOR'S ABSTRACT.

ROENTGENOGRAPHY OF LARYNX. R. WALDAPFEL, Monatschr. f. Ohrenh. **70**:918 (Aug.) 1937.

Waldapfel points out that the roentgenoscopic examination of the larynx is done almost exclusively by lateral exposures. The chief disadvantage of this method is that the two sides cover each other and cannot be separately visualized. An exposure in the anteroposterior direction fails to show details, because the dense shadow of the spinal column covers up the lesser density of the larynx. The only way to exclude the interfering spinal column is by introducing the film between the larynx and the spinal column, that is into the hypopharynx, and then making a frontal exposure. To accomplish this, superficial anesthesia of the pharyngeal mucous membrane is necessary. The author says that Réthi employed this method more than twenty years ago; however, it seems not to have found a wide application. Convinced of the great value of such roentgen exposures of the larynx, the author introduced Réthi's method into clinical practice. He does not describe the details of the technic but demonstrates the results obtained with the method. He shows that the method is helpful (1) in determining the downward extension of processes of the vocal cords, (2) for the after-examination in cases of laryngeal operation, (3) for the determination of the length and extension of laryngeal stenoses and (4) for physiologic studies. He reports a number of cases and shows reproductions of the roentgenograms. EDITOR'S ABSTRACT.

Nose

ACCESSORY NASAL SINUSITIS IN CHILDHOOD, WITH A RECORD OF BACTERIOLOGICAL EXAMINATIONS. J. CROOKS and A. G. SIGNY, Arch. Dis. Childhood **11**:281 (Dec.) 1936.

This is an exhaustive study of a hundred cases of sinusitis in children ranging in age from 2½ years to 12 years. The diagnosis was made on clinical manifestations, inspection of the nose and throat, roentgen study, transillumination, and puncture and aspiration. The maxillary antrum is more frequently the seat of disease than the other sinuses. The ethmoid sinuses are probably second.

Cooks and Signy believe that sinusitis is a common cause of inflammation of the adenoids and to a lesser degree of inflammation of the tonsils. The first step in the treatment of sinusitis is the treatment of the sinus itself and not the removal of tonsils and adenoids. If the latter are infected and fail to respond to such treatment, they should be removed. If there is a large pad of infected adenoids interfering with respiration, treatment of coexisting sinusitis is not likely to lead to a cure until the pad is removed.

KELLY, Milwaukee. [AM. J. DIS. CHILD.]

SIZE OF THE FRONTAL SINUSES IN ACUTE AND CHRONIC MAXILLARY SINUSITIS. ANNA GLUTZ, Ztschr. f. Hals-, Nasen- u. Ohrenh. **39**:496, 1936.

Glutz directs attention to a report by Kartagener and Ulrich in which those authors demonstrated that 39 of 70 patients with bronchiectasis had chronic maxillary sinusitis. This high percentage seems to indicate that the concurrence of the two processes is not entirely accidental. Moreover, a comparison of the cranial roentgenograms of patients with and without bronchiectasis revealed that patients with bronchiectasis more frequently had small frontal sinuses or had no frontal sinuses at all. In view of these observations, the concurrence of bronchiectasis and sinusitis has been regarded as a coordinated developmental inhibition or deformity on two different sites of the respiratory tract. The author decided to study the size of the frontal sinuses in patients with chronic maxillary sinusitis that does not concur with bronchiectasis. On the basis of 100 roentgenograms she demonstrated that in cases of chronic maxillary sinusitis the size of the frontal sinus does not differ from the normal. Accordingly, the size of the frontal sinus in patients with chronic maxillary sinusitis cannot serve as an indicator of a

constitutional anomaly, as is the case in maxillary sinusitis that concurs with bronchiectasis. As indicated by Kartagener and Ulrich, the latter form of sinusitis takes a special position among the chronic disorders of the accessory sinuses. The size of the accessory nasal sinuses seems to be of no importance in the pathogenesis of chronic maxillary sinusitis.

EDITOR'S ABSTRACT.

Miscellaneous

OTOGENOUS ABSCESS OF THE PARIETAL LOBE. CYRIL B. COURVILLE and J. M. NIELSEN, Bull. Los Angeles Neurol. Soc. **1**:65 (June) 1936.

Two cases of abscess of the parietal lobe secondary to homolateral otitis media are reported. In the first case a woman aged 60 who had had recurrent left hemiplegia presented deep coma, deviation of the eyes to the left, irregularity of the pupils, with the left larger than the right, flaccid left hemiplegia and a Babinski reaction bilaterally. The spinal fluid was opalescent, slightly xanthochromic and under elevated pressure. Autopsy revealed bronchopneumonia bilaterally, mucopurulent exudate in the left middle ear and an encapsulated abscess just under the cortex of the left parietal lobe. A thick greenish exudate was present in the leptomeninges over the abscess. A second, fresh abscess was observed anterior to and continuous with the first. Streptococci were cultured from the ear and from the abscess.

In the second case, a 4 year old Negro boy, who had always been mentally retarded, was suddenly paralyzed in the right arm, after which convulsions occurred in the right extremities and soon became generalized. The spinal fluid was under a pressure of 400 mm. of water and contained 44 cells per cubic millimeter. Signs of meningeal irritation followed. Autopsy revealed congenital pulmonary stenosis, pus in both middle ears and a large abscess in the left superior parietal lobule, which was poorly encapsulated and superficial. Streptococci were cultured from the ears and from the abscess.

Courville and Nielsen emphasize that the abscesses in these cases could not have arisen by direct extension, but are inclined to believe that they arose by retrograde extension along the cerebral venous channels. They point out that otitis media may not be recognized, even at autopsy, unless it is carefully sought and suggest that cerebral abscess of "unknown etiology" may often arise from a focus in the ear.

MACKAY, Chicago. [ARCH. NEUROL. & PSYCHIAT.]

GLOSSOPHARYNGEAL NEURALGIA. W. B. HOOVER and J. L. POPPEN, J. A. M. A. **107**:1015 (Sept. 26) 1936.

According to Hoover and Poppen, trigeminal and glossopharyngeal neuralgia are alike in all respects except the location of the agonizing flashes of pain and of the trigger areas which set off these paroxysms. The trigger areas in glossopharyngeal neuralgia include the pharyngeal wall, the tonsillar region, the base of the tongue and, rarely, the ear, while trigger areas in trigeminal neuralgia occur in the buccal mucous membrane and about the lips, nose and various areas of the face. When the first or second division of the trigeminal nerve is affected, there should be little or no difficulty in the differentiation of these two types of neuralgia, but when the third division is involved, more care must be exercised to differentiate this form from neuralgia of the ninth nerve. Cocainization of the mucous membranes over the distribution of the ninth nerve will, as a rule, control temporarily the paroxysmal pain from this nerve. Medical and surgical procedures are available in the treatment of glossopharyngeal neuralgia. In the authors' experience trichlorethylene has been the only drug really efficient in giving a marked amount of relief from this condition. It is administered by inhalation of from 15 to 30 drops three or four times a day. The surgical treatment of choice is the intracranial section of the ninth nerve in the posterior fossa.

EDITOR'S ABSTRACT. [ARCH. NEUROL. & PSYCHIAT.]

PROBLEMS OF LOCALIZATION WITHIN THE CENTRAL VESTIBULAR SYSTEM. R. LEIDLER, Monatschr. f. Ohrenh. **70**:176 (Feb.); 267 (March); 472 (April); 544 (May); 725 (June); 801 (July) 1936.

In this first series of articles on localization within the central vestibular system, Leidler takes up the inflammatory nonpurulent diseases of the brain stem. He gives his attention chiefly to two problems: (1) the relation between the slow and the rapid component of the vestibular nystagmus, and (2) the relation between the nystagmus after rotation and the nystagmus after calorization. In the introduction he says of the first problem that the dispute as to where the two components originate has already been answered, to the extent that for their elicitation the reflex apparatus (the labyrinth and its connections with the nuclei of the eye muscles) is adequate and higher portions of the brain (including the cerebellum) are not required. However, how and where within these limits the two components originate is still unknown. Of the second point he says that the inequality of the two reactions is already proved by the fact that rotation represents an adequate stimulus for the labyrinth, whereas the caloric stimulus is inadequate. This factor indicates that the genetic mechanisms of these two reactions cannot be identical. The author cites clinical observations that throw light on these problems. He cites the case histories of 15 patients who had various forms of nonpurulent encephalitis but who had in common disturbances in the central vestibular system. In all these cases only the vestibular apparatus was involved. The caloric test revealed entirely normal conditions in none of the cases; in the majority, severe disturbances existed, in several cases there being complete unresponsiveness on both sides. The author discusses this disturbance on the basis of experiments on reptiles, which seemed to indicate that the cold reaction is a much younger phylogenetically and consequently a more vulnerable reflex than the rotatory reflex. The impairment of the caloric stimulatability predominates greatly over that of the rotatory stimulatability. In all patients, with the exception of 2, the calorization and frequently also the examination in the rotation chair disclosed "abnormal reactions," chiefly of the eyes but in rare instances also of the head. These abnormal reactions consisted chiefly of deviations of the form of the nystagmus, which were due to disturbances in the relation of the slow to the rapid component in favor of the first, and in a lesser degree of disturbances in the rhythmic course of the nystagmus. The author describes analogous abnormal reactions in animals that had been operated on, in anencephalic human beings after birth injuries and in persons with disturbances in the consciousness. He reaches the conclusion that the disturbance of the rhythmic nystagmus, as the phylogenetically younger and probably also the functionally superordinated mechanism, leads to an "unlocking" of the phylogenetically older functionally subordinated mechanism of the monophasic deviation.

EDITOR'S ABSTRACT.

NEW FUNCTIONAL AND DEVELOPMENTAL CLASSIFICATION OF SKULL BONES: INFLUENCE OF THEIR STRUCTURE ON INFECTION. W. P. EAGLETON, Monatschr. f. Ohrenh. **70**:772 (July) 1936.

Eagleton first gives a historical survey of the surgical treatment of infection of the pyramidal apex and discusses its relation to meningitis. He points out that the apical suppuration is frequently considered from the same point of view as mastoiditis, namely, as if the suppuration were an infection of a pneumatic bone, whereas in reality it is an entirely different disorder, which must be estimated from a different surgical standpoint. Because in certain cases of apical suppuration there are recurrences of meningitis, the author decided to investigate the development of the bones. He emphasizes that the functional peculiarity of a tissue is dependent (1) on the embryonic layer from which it is derived and (2) on its localization within the animal organism. In order to clarify the function, the author classifies the bones of the neurocranium into four groups: (1) the bones of the cranial vault, which develop from membranes; (2) the bones of the sense

organs, which develop from cartilage; (3) the passive (pneumatic) protecting bones of the sense organs, which develop partly from cartilage and partly from membranes (mastoid process), and (4) active protecting bones of the cranial base, which contain red marrow and which have essentially the same structure as the other skeletal bones. In discussing the reactions of the different bones to infection, the author emphasizes that each of three types of bone (the pneumatic protecting bone of the mastoid process, the peculiarly constructed capsule of the internal ear and the marrow-filled petrosal apex) has, on the basis of its development, a specific histologic structure and a different reaction toward infection, the latter being determined by the stage of the growth of the bone at the time of infection. These two factors, the genesis of the bone and the stage of its development, exert an influence on the type and course of the infection. The author further gives his attention to the surgical aspects of the infection of the bones that contain marrow, giving especial attention to the function of the sphenoid body with its red marrow. He emphasizes that the body of the sphenoid bone is an important protecting organ for the cranial base; it protects especially the pons against infections. Further the author discusses the petrosal apex, particularly its capacity for defense against infection. As long as the apex contains fat, it is capable of metaplasia: that is, under the influence of an advancing infection, the fat can be transformed into red marrow, and if this is the case, the apex takes an active part in the combat of infection, a function which it has in common with all bones which contain primordial red marrow. In discussing the pneumatization of the petrosal apex, the author emphasizes that it differs essentially from the pneumatic growth of the mastoid process. Moreover, the pneumatization never spreads from the petrosal apex to the sphenoid body. In the conclusion the author expresses the opinion that the intensive nutrition of the sphenoid body and, to a lesser degree, of the petrosal apex by bilaterally arranged and free anastomosing blood vessels is the cause of the great reparatory capacity and the resistance to infection which characterize these bones.

EDITOR'S ABSTRACT.

Society Transactions

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

WALTER H. THEOBALD, M.D., *President*

October 4, 1937

THOMAS W. LEWIS, M.D., *Secretary*

USE OF SULFANILAMIDE IN THE TREATMENT OF STREPTOCOCCIC CARRIERS. Presented by DR. ARCHIBALD HOYNE and DR. HAYS BAILEY (by invitation).

Public health control of scarlet fever depends for the most part on the presence or absence of carriers of hemolytic streptococci in the population. In 1934 it was found that among 4,315 patients released from the Municipal Contagious Disease Hospital after having had scarlet fever in eighteen months there were 190 carriers known to be responsible for cases of secondary scarlet fever. Thus, it is apparent that any treatment that would eliminate the carrier state would be a marked advance in the control of the disease.

One year ago it was decided to test the efficiency of sulfanilamide because of the reports of its specific action for streptococci. The plan provided for administering the drug to patients with scarlet fever during the last quarter of the four week period of quarantine. Alternate patients were given one 5 grain (0.32 Gm.) tablet of sulfanilamide three times daily between meals. Cultures were made of material from the noses and throats of treated patients as well as of controls at the end of seven days. It was soon evident from examination of cultures that no influence was exerted on the presence of hemolytic streptococci, and the dose of sulfanilamide was therefore doubled, each patient receiving 30 grains (0.19 Gm.) daily.

Of 183 patients 58 served as controls, and 125 received the drug. Cultures of material from the nose and throat at the termination of quarantine gave negative results for but 7 of the controls and 36, or 20.8 per cent of the treated patients. The age distribution of the treated patients is interesting, patients under 7 years of age forming almost 50 per cent of the group. The importance of age as a factor in the control of scarlet fever has been previously referred to, and the removal of tonsils and adenoids before school age should play an important role in lowering the incidence. The largest percentage of negative results occurred in the group over 20 years of age (10 of 17 patients). It is the result in this group that makes the figures appear favorable for the drug.

Normally, without special treatment from 35 to 40 per cent of patients fail to harbor hemolytic streptococci at the end of four weeks. The apparently favorable results of treatment with sulfanilamide in this series of patients are undoubtedly due to the small number of patients studied. On the basis of the data presented it is concluded that, irrespective of any value sulfanilamide may have in the treatment of acute streptococcic diseases, it is not effective in the eradication of hemolytic streptococci from the noses and the throats of patients with scarlet fever at the end of a four week period of quarantine.

RESPONSE OF THE BLOOD CELLS TO SULFANILAMIDE THERAPY. Presented by DR. JOHN A. BIGLER, Highland Park, Ill. (by invitation).

Although sulfanilamide is widely used, little is known of its mode of action. Symptoms of toxicity, such as cutaneous rashes, nausea and vomiting, dizziness and hyperpyrexia, have been reported during administration of the drug. Cyanosis

during therapy has been mentioned. So far, a survey of the literature shows relatively few cases of depression of the number of blood cells, although there is an undercurrent of feeling that this medicament may cause the same marked depression of the blood elements as is being reported with such drugs as aminopyrine.

Careful observations have been made on 93 patients treated with sulfanilamide at the Children's Memorial Hospital. With the idea of determining the response of the leukocytes, daily blood counts and Schilling differential counts were made before, during and after such therapy for a group of 33 patients. The leukocyte counts were made at approximately the same hour of the day. A dose of 15 grains (0.16 Gm.) per 20 pounds (9 Kg.) of body weight was closely adhered to. Of the 33 patients, 27 had leukocytosis and 6 had a leukocyte count within normal limits when the therapy was started. In both groups there were patients with infection and fever, patients with infection but no fever (usually chronic infection) and patients with no evident infection.

1. *Response When Leukocytosis Was Present.*—(*a*) Infection with fever was present in 17 patients; in 7 a decrease of the leukocyte count to normal occurred in a crisis-like manner within twenty-four hours after the fever had subsided; in 4 the leukocyte count decreased in a lysis-like manner in from one to five days after subsidence of the fever; in 6 critically ill children who received this therapy for only a short period there was no change.

(*b*) Three patients in whom an infection without fever was present had leukocytosis; in 2 the leukocyte count decreased to normal in a crisis-like manner; the third showed a decrease to normal in four days.

(*c*) Leukocytosis was present in 7 patients in whom there was no evident infection; 4 of them had recently recovered from an infection of the throat. In 4 of the 7 patients a crisis-like drop to normal occurred within from twenty-four to thirty-six hours after medication was started; in 2 a lysis-like decrease took place in three days. One patient with between 8,000 and 10,000 leukocytes showed no change in the number of white cells after five days.

2. *Response When the Leukocyte Count Was Normal.*—(*a*) The 1 patient who had an infection accompanied with fever was given sulfanilamide for two days, when the therapy was discontinued because of vomiting and severe acidosis. There was no therapeutic effect in this instance.

(*b*) In 2 of 3 patients who had infection without fever the leukocyte count decreased to 4,800 and rose again when the drug was discontinued. These were the only 2 patients in whom leukopenia developed. In the third patient there was no change from normal.

(*c*) No infection was present in 2 patients with normal white cell counts. In neither was there any change in the leukocyte count during or after therapy.

Besides 6 patients to whom the drug was not administered until the infection subsided, there were 21 patients with leukocytosis. In 13 the number of leukocytes decreased to normal in a crisis-like manner within from twenty-four to thirty-six hours after subsidence of the fever. In 7 it returned to normal in a lysis-like manner only after forty-eight hours or more, while in 1 there was no change. Of 6 patients with a normal white cell count, moderate leukopenia without neutropenia developed in 2; in the other 4 the leukocyte count did not change.

The leukocytic response was in the nature of a marked absolute reduction of all types of cells, without any characteristic relative change in their proportions, according to the Schilling count. An increase in the leukocyte count which could be attributed to sulfanilamide did not occur in any case. It did seem, however, that the drug causes a depression of the number of white blood cells and this was borne out by 2 cases in which leukopenia developed and by the spectacularly rapid decrease in the leukocytes as the infection subsided. It seems that the value of sulfanilamide is due to its own action in the blood stream rather than to its

enhancing the power or increasing the number of the leukocytes so that they might control the infection.

In compiling the statistics embodied in this study, I took a conservative attitude. Consideration was given not only to the individual case but to similar cases being observed at the same time in which sulfanilamide therapy was not employed.

It is concluded that sulfanilamide is an effective therapeutic agent for most infections with the hemolytic streptococcus. Cyanosis is frequent during administration of the drug. There is evidence that sulfanilamide has a tendency to cause a depression of the leukocyte count. The action of the drug seems to be independent of the leukocytes, in that it does not produce an increase in the total number or in the proportion of polymorphonuclear cells. From this study it is apparent that frequent blood counts should accompany sulfanilamide therapy. My associates and I have encountered 2 more cases of leukopenia recently in patients with infection, fever and leukocytosis during sulfanilamide therapy.

SULFANILAMIDE THERAPY. Presented by DR. GEORGE SHAYNIN LIVINGSTON.

This paper analyzes three phases of sulfanilamide therapy: evidence of the clinical value of the drug, especially in otolaryngology; the method of its administration, and its untoward and dangerous effects.

Most of the material available for study deals with infections caused by beta hemolytic streptococci. Sulfanilamide has been used in treating other types of infections with varying results. Certain criteria must be established before judgment can be passed. In the large group of infections which usually terminate favorably, individual cases are valueless. If large series consistently show more rapid resolution, shorter and milder course and fewer complications than previous experience has taught practitioners to expect, a strong case in favor of sulfanilamide therapy will be established. Reports already published are sufficiently favorable to encourage continued large scale study. With the smaller group of infections which have a high mortality, such as streptococcic meningitis, a significant reduction in the death rate establishes the value of the drug much more definitely. The mortality from streptococcic meningitis has apparently been markedly reduced, and it seems probable that future reports will be even more favorable, owing to more widespread use, earlier administration and a better knowledge of adequate dosage.

Five cases in which sulfanilamide was employed are presented from the Children's Memorial Hospital. These are typical of a large group in which a diagnosis of surgical mastoiditis was made and in which, according to the usual standards, operation would have been performed. All 5 patients recovered promptly after administration of sulfanilamide. Cases in which parallel results could have been expected without the use of the drug are omitted. In viewing the entire series of hospitalized patients with otitis media and mastoiditis, one has the feeling that a significant advance has been made.

Not all the patients treated with this drug were benefited. Cases in which treatment was unsuccessful divide themselves into two groups. In the first inadequate doses were given; less than the suggested amounts were used and administration was stopped too soon. The second group comprises 7 cases of acute suppurative otitis media in which proper administration of sulfanilamide seemed to exert no beneficial effect. At present the chief value of the drug seems to be against infections caused by the hemolytic streptococcus, the meningococcus and the gonococcus; Streptococcus viridans and Staphylococus aureus seem to be resistant to its action.

The mode of action of sulfanilamide has not been determined. It is probable that it retards multiplication of the organisms sufficiently to permit the normal defense mechanisms of the body to destroy them. Satisfactory action depends on the continued presence in the body of an adequate concentration of the drug. Dosage will vary according to the rate of absorption and excretion.

Many derivatives of sulfanilamide and related compounds are being tested experimentally, but most of those reported on so far have proved to be either more

toxic or less effective than sulfanilamide. It seems wisest to use only sulfanilamide until some of the newer compounds have been proved conclusively to be superior to it.

From personal experience and from a review of the literature to date it is concluded that sulfanilamide has already proved valuable in the treatment of various infections, chiefly those caused by the beta hemolytic streptococcus. Its effectiveness varies with the subject, with the stage of illness in which treatment is begun and with dosage. Certain unfavorable effects have been noted, most of which disappear quickly when the drug is withdrawn. Of graver significance are reports of methemoglobinemia, sulfhemoglobinemia, acidosis, acute hemolytic anemia, agranulocytosis and optic neuritis. Frequent careful studies of the blood are of paramount importance. Finally, sulfanilamide should be considered as a valuable adjunct and not a panacea. All previously accepted means of medical and surgical treatment must continue to be used as indications arise.

DISCUSSION

DR. HOWARD C. BALLENGER: These reports have been confined largely to children. It is possible that in adults the picture may be different in certain instances if this drug is used extensively. About two weeks ago I was called in consultation by a general physician concerning a rather obese woman of about 50 who had acute nasopharyngitis. The culture showed hemolytic streptococci. The blood count was about 8,500. She got along nicely, except for the sore throat, which persisted. The physician gave sulfanilamide in the usual dose for three days. At the expiration of three days the blood count was 2,500. At this time, when I saw the patient, there was a dark spot on one tonsil about the size of a penny. This spot was later covered by a membrane. It was the typical bluish black necrotic area, characteristic of early agranulocytosis. Next morning the blood count was 2,000; on the opposite tonsil there was a similar area about the size of a pea, and there was a membrane on the uvula and the first tonsil. The next day the blood count was 1,000 and later dropped to 500, when death occurred. Whether sulfanilamide was a factor in this case I do not know, but it could have been.

The drug should not be used indiscriminately in the treatment of mild infections of the throat, especially in adults.

DR. ALFRED LEWY: Practitioners need to know more at present about the toxic effects. There is no way—no easy way, at least—of determining whether the course of the disease or the drug is producing the symptoms under discussion. I shall limit my remarks entirely to the treatment of meningitis and other intracranial consequences of otitis media. As chairman of the committee on meningitis for the Chicago area, last May I reported to the American Otological Society 8 recoveries from hemolytic streptococcus meningitis in cases in which cultures of the spinal fluid had given positive results. Eight patients recovered and there were a number who did not recover. In 6 cases of hemolytic streptococcus meningitis at the Cook County Hospital there were 4 recoveries, and I had personal knowledge at that time of 4 other cases at two other hospitals in which there was recovery. Dr. James G. Dwyer, chairman of the New York committee on otitic meningitis, reported fourteen cases in which recovery occurred. I do not know how many cases he knew of in which recovery did not occur. In view of the 97 + per cent mortality heretofore, this is a satisfactory showing.

In addition to these cases, I should like to mention one I observed recently, that of a 13 year old child who had a mastoid operation in May and went home apparently recovered. She returned to the hospital in August, having had a dizzy spell ten days before. Two days before entrance into the hospital she went into a coma, from which she had recovered at the time she entered the hospital. At the time of entrance she had fully developed meningitis and complete paralysis of the muscles of one eye and of the external rectus muscle of the other

eye. Hemolytic streptococci were recovered from the blood stream and from the spinal fluid on four different occasions. She recovered. I do not know what will happen to her in the next year.

I should like to mention 3 other cases. In all 3 a mastoid operation had been performed from ten days to four weeks before I saw the patient. In one the clinical symptoms of meningitis had been present at admission, and the condition proved to be sinus thrombosis. A week later severe occipital headache developed. The condition was diagnosed as a lesion, possibly an abscess of the posterior fossa, but while under observation the child recovered. The second case was almost identical except for the sinus thrombosis, and in that also, while I was watching and waiting, there was recovery. In the third case there was a choked disk, and roentgenographic study showed a definite abscess of the petrosa. Because of the absence of any other symptoms I did some watchful waiting, and the patient is apparently well. I do not know how long the patients will remain well. In all these cases the disease has occurred in the last six months. In all sulfanilamide was administered. I have a considerable list of recoveries without operation in cases in which sulfanilamide was used. These have justified a little watchful waiting, and in that respect some accepted surgical indications may have to be revised.

I have had no experience with intraspinal administration of sulfanilamide. I have depended entirely on the use of the drug parenterally or by mouth.

DR. GEORGE E. SHAMBAUGH JR.: I should like to report briefly 1 case. My patient had acute otitis media on December 4, with marked tenderness over the mastoid and a temperature of 103 F.

Three weeks later tenderness over the mastoid was still present, with profuse discharge from the ear. For one week there had been increasing pain in the right eye. The diagnosis was early coalescent mastoiditis, with probable infection of the petrous apex. Simple mastoidectomy showed an extensively pneumatized mastoid process. A week after operation paralysis of the sixth nerve developed on the same side. The pain behind the eye and the paralysis of the sixth nerve persisted, with profuse discharge from the middle ear and the roentgenologic picture of coalescent petrositis. Because the patient's general condition was good and the symptoms showed a tendency to gradual improvement, operation on the petrous apex was deferred.

Four months after the patient was first seen there was still a profuse discharge from the middle ear, the mastoid incision having healed within a few weeks of the operation. The paralysis of the sixth nerve and the pain behind the eye had cleared, but the roentgenologic picture continued to show a large area of cellular destruction in the petrous apex. Administration of sulfanilamide, 10 grains (0.65 Gm.) three times a day, was begun. Within twenty-four hours the profuse discharge from the ear had stopped, and except for a trace of moisture once or twice for the next few days, the ear did not discharge again. The hearing, which had been markedly diminished in this ear for four months, was normal two weeks later.

DR. T. C. GALLOWAY: All the cases of hemolytic streptococcus meningitis in which recovery has followed the use of sulfanilamide should be reported, because such data are more conclusive than the best possible laboratory test. I have never observed recovery from this disease before this year, but since January I have seen 2 patients of my own and 3 at the Cook County Hospital get well and have heard of 4 other patients who have recovered in the Chicago area. In 1 case of mine in which there were marked meningeal symptoms, a high cell count and many organisms, sulfanilamide was administered and a simple mastoid operation performed, and the postoperative course was as normal as that following any mastoid operation. In another case, in which the condition was secondary to labyrinthitis, an operation was performed on the labyrinth, with wide dural exposure, and sulfanilamide produced satisfactory results. When administration of the drug was stopped after two weeks, there was recurrence, and the condition was controlled again by the drug. Recovery followed.

In 2 cases in which there were definite indications for mastoidectomy the condition cleared in a few days, and the ears remained dry. Twice in cases in which the drug did not produce clearing I was struck by the spottiness of the mastoid —as if there had been healing when necrosis was not already advanced. It will be interesting to find out just how much of the pathologic picture can be destroyed if this drug can eliminate the infection.

The drug is new. The members will all remember that dinitrophenol was hailed as a wonderful cure. I saw a 27 year old woman who took it two years ago, and there were no symptoms until a few weeks ago, when she noticed failing vision. Now she has rapidly advancing cataracts. Because all the bad effects of sulfanilamide, especially the late effects, cannot be known, I think it would be a mistake to use it indiscriminately. It should be saved for the cases in which ordinary methods do not suffice.

DR. MELVIN REESE GUTTMAN: I should like to place on record what I believe to be the first case of cure of Streptococcus haemolyticus meningitis with sulfanilamide in the Chicago area. Last March there was admitted to the service of Dr. Robert Sonnenschein in the Michael Reese Hospital a girl 3 years of age who had had a running ear and intermittent fever for five weeks. Two days prior to admission she became much worse, and on that morning she had gone into coma. She was seen at 4:00 o'clock in coma, with a stiff neck and other classic signs of meningitis. Spinal puncture showed streptococci by smear and several thousand white cells. Culture showed hemolytic streptococci later. I performed an extensive mastoidectomy on the patient at 6:00 o'clock that evening. The dura was exposed and showed nothing; the lateral sinus was apparently normal. That evening I attempted to get some sulfanilamide. Unfortunately there was none available, but a supply came by airplane twenty-four hours later. The girl was still in coma and received a full dose that evening. She received large amounts of the drug for three days and came out of the coma. The temperature dropped from 105 to 99 F., and she made an uneventful recovery.

DR. AUSTIN A. HAYDEN: This is a timely, interesting and important subject; so important that publication of the program of the American Medical Association at Atlantic City, N. J., was held up ten days last year because of some investigation by Dr. H. F. Helmholz at Rochester, Minn. Many interesting things can be said about sulfanilamide. I have been informed by Dr. Paul N. Leech, of the Council on Pharmacy and Chemistry of the American Medical Association, that sulfanilamide is an old drug. It is the amide of sulfuric acid and has been known from the sixties or the seventies. It was first made in Germany as a dye, for which purpose it was not found to be satisfactory. Unsatisfactory dyes are frequently turned to medical use. For some reason that amide was neglected for all these years, until about 1931, when some experimental work on peritonitis in rats suggested its possibility.

I was impressed by a statement made by Dr. Leech in connection with the extent to which a drug of this sort will be used, not only because of its worth but because of the publicity, that a single drug house shipped to South America in August 2 tons of sulfanilamide. The price has been enormously reduced, in which connection it may be remembered that the first acetylsalicylic acid in the United States cost $5 a grain.

It is well to bear in mind that the name accepted by the Council on Pharmacy and Chemistry is sulfanilamide, not para-aminobenzene sulfonamide. I gather from the statistics presented on the board tonight that this drug has a specific action, but not in bony lesions, except perhaps in the bones of the head. Dr. Bailey showed the effect was nil in osteomyelitis of the tibia. My own experience has not been satisfactory, but the number of cases I have observed is limited, and in one case in particular in which I expected marvelous results death was prompt. The patient, however, was practically moribund at the time medication was begun.

I agree heartily that great care should be used. I fear that with the startling reports of successful administration that have been made administration of the drug may become a matter of over the counter drugstore prescription.

Dr. William James Noonan: The patient I saw had hemolytic streptococci in the spinal fluid and rigidity of the neck and was given a derivative of sulfanilamide known as prontosil (the disodium salt of 4-sulfamidophenyl-2′-azo-7′-acetylamino-1′-hydroxynaphthalene-3′, 6′-disulfonic acid) intramuscularly, sufanilamide by mouth and an 0.8 per cent solution of crystalline sulfanilamide intraspinally.

Dr. Samuel J. Pearlman: There have been recently observed at the Michael Reese Hospital 3 cases of Streptococcus haemolyticus meningitis, in all of which there were organisms in the spinal fluid. All the patients got well with sulfanilamide therapy.

I should like to add my bit to Dr. Galloway's remarks about dinitrophenol. I, too, know of a case in which the patient took the normal dose for two days and then almost two years later had characteristic dinitrophenol cataract. It is proper, in my mind, to use sulfanilamide for Streptococcus haemolyticus meningitis. No fair-minded person will deny that there has been a great advance in the therapy of that disease. This evening there have been more reports of recoveries in the past year than in many years preceding. I am not prepared, however, to accept fully the glowing reports of the efficacy of sulfanilamide against tonsillitis and mastoiditis. I believe that there is not one of the members who cannot match the cases mentioned tonight with similar results in instances in which sulfanilamide was not used.

Every one knows of the child or infant who has a high temperature due to acute otitis media; paracentesis is done; the ear discharges for a few hours; the temperature drops, and the patient is well. Every one knows about the instance of mastoiditis in which preparations for operation are all made but for some reason a short postponement is necessary and, to the amazement of all, spontaneous recovery occurs.

With a drug such as sulfanilamide, of which the late results are not known and for which each week brings more reports of unfavorable reactions, one should keep an open mind, use it intensively and with a free conscience only in those cases in which the condition is known to be usually fatal and in which much may be gained by therapy; lastly, one should avoid giving it credit for glowing recoveries from diseases which run a self-limited course.

Dr. George Thomas Jordan: Apparently sufficient information about this drug is available to justify going ahead and using it. Care is necessary, however. I have had 3 cases in which I thought I had to do a mastoidectomy—cases in which the cardinal symptoms were present and of long duration. In those 3 cases prontosil was given hypodermically, and recovery followed. I had a case which parallels that of Dr. Shambaugh, the condition following scarlet fever. Bilateral mastoidectomy was performed. Trouble persisted on the right side. By the time I wanted to operate again, the child had chickenpox. That child got well enough to go home, and the infection of the ear started up again, accompanied with paralysis of the right external rectus muscle and pain behind the eye. The mastoid was reopened, and the child recovered and has been well for three months. I think it is possible to have sufficient confidence in this drug to use it, if it is used judiciously.

Dr. Paul H. Holinger (by invitation): There was a similar case in the medical service of the Research and Educational Hospital last March. The patient had an acute severe streptococcic infection of the lung. Bronchoscopy revealed the process to be limited to the posterior division of the lower lobe of the left lung, and culture of the secretion aspirated from this area showed hemolytic streptococci. There was associated low grade nephritis, with occasional red cells and casts. The administration of sulfanilamide was followed by an acute exacerbation of the urinary changes, with a shower of red cells and casts. In view of subsequent work, as well as the data supplied by Dr. Livingston the regular dose of the drug probably should not have been given. Its administration was discontinued entirely, however, and the renal flare-up subsided.

DR. HARRY L. POLLOCK: I have not much to report in the way of meningitis. Regarding infections of the throat, I have had two cases recently, one in a young man of 27, with a typical streptococcic sore throat. The culture showed hemolytic streptococci, and the temperature was 105 F. on the first day and 105.6 F. on the second day. I started that night with the drug, and within twelve hours the patient's temperature dropped to about 99 F. The man was very sick—so sick that an internist was called. The temperature went up to 102 F. next day, and the dose was increased a little. His temperature was normal that evening and has been normal ever since.

I have had 2 cases of streptococcic sore throat, mostly in the pharynx. In both the temperature dropped within twelve hours and the patient made a rapid and uneventful recovery. I feel that this drug should be used carefully; but there are conditions in which it is indicated, and if the patient is watched carefully I think any danger can be avoided. The young man I spoke of was cyanotic before the drug was given; the temperature was 105.6 F., and within twelve hours the cyanosis disappeared and there was no further trouble.

DR. JOHN A. BIGLER, Highland Park, Ill. (by invitation): I should like to say that since I have been interested in the response of the blood cells during sulfanilamide therapy I have talked to many persons. One physician said he had a case in which agranulocytosis developed as a result of the use of the drug. When the patient died leukemia was found. I think one should be careful in describing changes in the blood, because blood dyscrasia may develop as a result of the disease rather than of any therapy.

DR. GEORGE SHAYNIN LIVINGSTON: I want to say to Dr. Lewy that I do not disagree with his contention that surgical indications may have to be revised. I think the accepted indications have been definitely changed by cases in which the patient seemed entirely ready for surgical intervention and did not receive it. By my statement I meant that in spite of treatment mastoidectomy may still be required. With streptococcic meningitis the patient cannot get any worse, but in cases in which some means of helping the patient other than the drug is available I feel that it is only fair to use such means. Nevertheless, if in spite of treatment operation seems indicated, it should be performed. The danger is not so much with otologists as perhaps with other practitioners, not specialists, who have a patient with aural disease and may, hoping against hope, administer sulfanilamide. I have some figures which I omitted from my talk before because I did not feel they properly could be included in a statistical group. In the nine months ending Sept. 30, 1937, I performed 22 mastoidectomies for mastoiditis; in the same period of 1936 I performed 59, and in the same period of 1935, 76. I do not offer those figures as proof that sulfanilamide has reduced the number of mastoidectomies, although I feel it had something to do with it. I do not feel that the figure is large enough to justify drawing any conclusions. It is known that in some years the respiratory organisms in epidemic form are much more severe and virulent than in other years. In the city clinics the general economic conditions must be borne in mind. These may have some bearing on the number of people who come to the clinics. There are fewer now than there were three or four years ago—a possible factor, other than sulfanilamide, in the total number of cases and of operations.

The criticism Dr. Pearlman made, I think is entirely valid. I tried to be careful to eliminate the type of case he mentioned, and I realize that a few I put in were not above criticism, because the members all know of cases in which operation was imminent and was postponed for some reason, and the patient got well without sulfanilamide. That sequence of events can be applied as a criterion only in large groups of cases. If large clinics will continue to record their experiences year after year, perhaps in three or four years there will be such a body of evidence that the value of the drug will be incontrovertible, but if a case here and there is saved it will not mean anything.

WALTER H. THEOBALD, M.D., *President*

Nov. 1, 1937

THOMAS W. LEWIS, M.D., *Secretary*

EXPERIMENTS ON THE CONDUCTION OF SOUND IN THE EAR. Presented by DR. H. G. KOBRAK (by invitation).

The acoustic reflex of the muscles of the middle ear is demonstrated. The close relationship between acoustic stimulus and muscular contraction is shown. Each tone produces a definite contraction according to its frequency and intensity. High and low tones have a higher threshold than the middle range tones, and the muscular contractions produce a curve which resembles that of the threshold of hearing. In regard to intensity, three groups of stimuli can be distinguished: first, underthreshold stimuli for both muscles; second, a narrow range of intensities which are overthreshold for the stapedius, and, third, overthreshold stimuli for both muscles.

The mechanical effect of both muscles consists in a diminution of the vibration of the ossicles. The tensor muscle works mainly on the lateral ossicle, the hammer, while the effect of the stapedius is most outspoken on the stapes. The muscular contractions change the sensitivity of the ossicular chain. The conditions of sound conduction are therefore not constant. Each tone travels over an ossicular chain the sensitivity of which is specially adapted to the physical qualities of the stimulus.

The fidelity of sound recording is discussed. The middle ear is described as a sound recorder the resonance frequency of which is situated in the region of important speech frequencies. The construction of artificial sound recorders (e. g., microphones) is discussed and compared with that of the ear. The fidelity of the ossicular chain is rather poor. Frequencies of the resonance range are "overrecorded" beyond their true value. Weak stimuli are therefore amplified and underthreshold tones perhaps made audible. The sensitivity of the ossicular chain is enhanced in the resonance zone. Loud tones which need no amplification are cut down by the muscular reflex. In this case the overrecording due to resonance is diminished by the muscular contractions. The reflex acts as a correcting device.

DISCUSSION

DR. JOHN RALSTON LINDSAY: The work which Dr. Kobrak has just presented demonstrates the way in which the intrinsic muscles of the ear function and the effect which they exert on the conduction of sound waves to the inner ear.

The relation between the muscular contraction and the sensation of hearing has been definitely established by observations on human beings, in over thirty of whom it has been possible to observe the stapedius reflex.

These preliminary observations have laid the foundation for further investigations along two entirely different lines:

1. The effect which these muscles have on the hearing under various pathologic conditions. As Dr. Kobrak has said, the effect of the muscles has been neglected, and information up to the present has been largely theoretic. Investigation along this line is under way.

2. The reflex response of these muscles to sound. This provides an objective measure of cochlear function in the animal and opens up an avenue for experimentation on hearing.

The muscular reflex of the middle ear involves the auditory tract only as high as the olivary nucleus and is therefore not affected by the higher centers. It therefore provides an ideal means of studying such questions as auditory fatigue and the effect of masking. The reflex provides a relatively simple method of testing an animal's hearing when compared with the specialized technic necessary to condition animals or with the complicated electrical equipment necessary to

obtain the electrical responses. The factors of attention and fatigue of higher centers are eliminated. Concerning the electrical method, it may be said that until more is known about the origin of the electrical response it should not be assumed that that is an index of hearing under all circumstances.

DR. ALFRED LEWY: I should like to ask Dr. Kobrak at what frequencies he gets the reaction and Dr. Lindsay how he determines at what level in the brain that reflex operates. Were sections made? Does Dr. Kobrak know of any experiments on the ears of patients who had had resection of the gasserian ganglion?

DR. EDWIN JEAN BLONDER: I should like to know how the stimulation is localized to the olivary nucleus. Is it possible to cut the olivary nucleus, or is a complete section made? There are some pathways from the cochlear nuclei to the olivary nucleus, and I wonder whether the presenter's method possibly injures some of these tracts before the olivary nucleus is reached.

DR. T. C. GALLOWAY: It must be obvious that the tone is heard without activity of the reflex, because the tone must precede the reflex and activate it. Is the reflex then essential to hearing?

DR. GEORGE JAMES DENNIS: Will Dr. Kobrak describe the pathway of the reflex?

DR. H. G. KOBRAK (by invitation): In reply to the question of Dr. Lewy, the curve of the muscular contractions resembles that of the threshold of hearing. In the rabbit the optimum of the muscular reflex has been found at about 2,000 double vibrations. Unfortunately, the optimum of hearing in the rabbit is not known. In the human being the optimum of muscular contractions was determined to be about 1,500 double vibrations. In patients with pathologic conditions in whom the stapedius muscle was observed through a perforation, the resonance frequency may be changed. The influence of a pathologic resonance frequency on hearing has never been studied. Dr. Lewy asked whether a patient whose gasserian ganglion has been extirpated has been subjected to hearing tests. No. However, there has been an investigation on patients with facial paralysis. In such cases the branch of the facial nerve to the stapedius muscle was thought to be paralyzed. The hearing was found to be impaired. I concluded that my findings were a proof for the accommodation theory, since the loss of the muscle diminishes hearing. However, these conclusions are not definite.

In reply to Dr. Blonder as to the influence of the olivary nucleus on the reflex, the central part of the reflex has been studied by Hammerschlag. By cutting experiments it was established that the trigeminal and the facial nucleus are connected with the primary acoustic nucleus. Sections above those nuclei leave the reflex intact. So far as I know, the olivary nucleus has not been mentioned in the literature as an essential part of the reflex arch.

In reply to Dr. Galloway, the effect of the muscular reflex cannot be determined by threshold tests. In obtaining our typical audiometer curves we use stimuli which are below the threshold of the muscular contractions. There may be some influence of the muscles on the loudness of overthreshold stimuli.

CONTROL OF DELAYED HEMORRHAGE FOLLOWING TONSILLECTOMY. Presented by DR. WILLIAM ALLEN SMILEY.

Otolaryngologists have spent considerable thought and anxiety over the possibility of hemorrhage following tonsillectomy. Most articles on the subject are concerned with control of hemorrhage at the time of operation or a few hours later and many ingenious devices have been provided for the application of pressure and the placing of sutures or ligatures, while the virtues of astringents, caustics and hemostatics have been extolled.

Delayed or secondary hemorrhage has been mentioned occasionally as a possibility, and a few cases have been reported of hemorrhage starting between the fourth and the fourteenth day, in one recurring for twenty days. These hemor-

rhages occur mostly in adults, are more frequent after local than after general anesthesia and are usually unilateral. They occur after about 2 per cent of adult tonsillectomies, usually between the fourth and the seventh day and usually in the early morning. The amount of blood lost may be only a few cubic centimeters, or the patient may vomit several hundred cubic centimeters of blood and gastric contents.

In treatment of these hemorrhages the clot which is usually present in the tonsillar fossa is removed and the bleeding point visualized. In a series of 29 cases in the past five years the bleeding has been controlled by the superficial injection of a 1 per cent solution of procaine hydrochloride around the bleeding point. The amount of procaine hydrochloride used has never exceeded 1.5 cc. The action is explained thus: The fascia of the tonsil bed is slightly elevated by the injection, causing the bleeding vessel to retract below the surface. The bleeding stops promptly. In 3 instances physiologic solution of sodium chloride was substituted with satisfactory results, but 1 of the patients complained of considerable pain immediately following the injection.

Five patients in this group had failed to respond to ordinary methods of treatment, such as the removal of the clot, the administration of ice pellets, the application of an ice bag to the throat and the use of astringents or even of caustics, and the bleeding had continued or recurred for over two hours. In no patient was there a return of bleeding. There was no resulting infection, and the throat was less sore than after the use of astringents or caustics.

DISCUSSION

DR. FRANCIS L. LEDERER: I cannot disagree with Dr. Smiley. He has positive evidence from 29 cases. As to injecting or introducing a needle, where is he going to introduce it? He will go through an area of infection. I wonder if there is any thought in his mind of producing a parapharyngeal abscess as a result of injecting procaine hydrochloride or any other solution into these tissues, not through lack of sterility of the solution but through the introduction of organisms from the outside. That might be borne in mind when one is considerng this method as a panacea. It is hard to visualize the mechanism, namely, edema producing retraction of or pressure on a vessel, although I have no better explanation for its action. There is epinephrine in the solution. Perhaps that accounts for the momentary hemostasis. Were there any recurrences of bleeding?

DR. AUSTIN A. HAYDEN: I remember well when Dr. Beck first brought out control of hemorrhage in tonsillectomy by suturing a piece of gauze into the tonsillar fossa. I think that was about 1912, perhaps a little earlier. The bleeding point should always be looked for and will usually be found. It seems to me that this is a primary consideration in any event, if hemorrhage is to be controlled by injection, suture or what not. I agree with Dr. Smiley that pressure can stop that and that the injection of any fluid, whether it is physiologic solution of sodium chloride or an antiseptic, can conceivably stop a hemorrhage, although I have never used that method. I always put a suture in around the bleeding point or bleeding area. I should be much afraid of doing what Dr. Smiley has suggested, because I think it would mean another call later in the night.

DR. JOSEPH C. BECK: In discussing Dr. Smiley's paper, I wish to say that the number of secondary hemorrhages with tonsillectomy that he reports is startling to me. I have removed a great many tonsils in my day, but I do not believe I can record 3 cases of genuine secondary bleeding—that is, if I understand Dr. Smiley's differentiation between a septic condition of the throat and bleeding that cannot be stopped because the blood vessels cannot retract. This method seems perfectly feasible if Dr. Smiley can avoid what Dr. Lederer has spoken of, namely, injecting infective material into a sterile field. However, I can readily see that injecting a large quantity of liquid into the tissues about the bleeding point can cause a compression which may be sufficient to get a reaction that might close it. If there is a great deal of elevation, so that the loose tissue contains the vessel

below, it seems to me that hematoma might develop. Suturing a septic field—attempting to tie a vessel—has not been successful in my hands, as the suture tears out. I think Dr. Smiley's method, which is simple, may produce some good results.

DR. P. W. GOOD, Kenosha, Wis. (by invitation): I have been unfortunate in having secondary hemorrhage in about 10 cases even though I have not done many tonsillectomies. In all cases it has seemed to me to be bleeding from the paratonsillar vein, which runs vertically in the tonsillar fossa. For secondary hemorrhage, I believe that the vein must be left intact at the time of the operation and that the wall of the vein sloughs through on the sixth or the seventh day. Often the bleeding is of short duration and stops if the patient sits up and holds a piece of ice in his mouth. In 1 case it was thought best to suture the bleeding point during general anesthesia. The easiest and surest way that I have found for doing this is with a double-armed sponge holder so made that gentle pressure is exerted on and between the tonsillar fossae. The patient is usually prepared with 0.25 grain (0.016 Gm.) of morphine. I am much interested, however, in Dr. Smiley's method and will try it if I am unfortunate again.

DR. SAMUEL SALINGER: I should like to know on whose authority it was determined that a tonsil bed was a septic field. When the tonsils are removed, a clean wound covered with a fibrincus exudate remains, and it never has appeared to me to be septic. It is true that a large number of saprophytic bacteria may find their abode in that membrane, but they are not dangerous. If the field were septic, every time a physician put a suture into the tissues he would have infection; certainly there would be more instances than have been reported. I say this to support Dr. Smiley's statement that the mere introduction of a needle into the average tonsil bed is not likely to carry infection.

DR. LEOPOLD BENNO BERNHEIMER: I recall an instance in which infection did play an important role in the production of secondary tonsillar hemorrhage. The patient was a man of about 18 years who had profuse hemorrhage eighteen hours after tonsillectomy. Examination revealed a profuse oozing from the right tonsillar fossa, with a dissecting hematoma extending down the fascial planes of the neck over the carotid sheath. A decision to ligate the external carotid artery was immediately made. In exposing the vessel for ligation, actual free blood and blood clots were encountered, burrowing down between the superficial and the investing layers of the deep cervical fasciae. After ligation of the external carotid artery, hemorrhage was controlled for about seventy-two hours, at the end of which time it recurred. The boy at this time showed clinical evidences of pharyngomaxillary infection and was almost exsanguinated. The wound in the neck was reopened, and a pharyngomaxillary abscess was found to have eroded the internal carotid artery. The common carotid artery was then ligated, and after repeated transfusions the patient made an uneventful recovery.

DR. ROBERT HOSEA GOOD: I think it would be a mistake to consider every secondary hemorrhage infectious. Often the tonsillar cavity on the sixth or the eighth day can be seen to contain remnants of dry membrane, and when these fall off a blood vessel may be opened and hemorrhage produced. The only infections I have seen are in cases in which a blood clot is allowed to remain for a number of days in the tonsillar cavity.

DR. WILLIAM ALLEN SMILEY: I have not encountered any cases of general oozing from the tonsillar fossa in which I was not able to visualize the point from which the hemorrhage was originating.

With regard to Dr. Lederer's question, the point of injection has been a small distance, maybe 0.5 cm., from the bleeding point, and the needle has been introduced only about 0.25 cm. The amount of fluid injected has not, as I mentioned, been more than 1.5 cc. in any patient. The first time I used the treatment was in preparation for a suture. When I put in the anesthetic the bleeding stopped, and I waited, but it did not start again. I do not feel that there is any more danger in introducing a hypodermic needle into the tonsillar fossa than in introducing

a needle with a suture attached. I have not used any clamp, either a double-inside or an inside-outside clamp, because I figured the discomfort to the patient was such that I preferred to avoid it if possible. M. C. Myerson (Hemorrhage Following Tonsillectomy, *Am. J. Surg.* **36**:151 [April] 1937) attributed little importance to infection in the tonsillar fossa as an etiologic factor in delayed hemorrhage. With regard to Dr. Bernheimer's case, I do not think I should be interested in trying procaine hydrochloride in such a case.

POSTOPERATIVE REPAIR OF THE PARANASAL SINUSES: EXPERIMENTAL STUDIES AND THEIR PRACTICAL APPLICATION. Presented by DR. MORRIS A. GLATT.

This paper will be published in full in a subsequent issue of the ARCHIVES.

DISCUSSION

DR. JOSEPH C. BECK: Dr. Glatt allowed me to study the preparations he has presented on the screen and to read his paper. He did not have time to read an important part of it, the review of the literature, which would have helped a great deal in understanding many points made. I should like to carry the members back a little in the history of disease of the sinuses. Hajeck and Jansen in Vienna, Austria, and Berlin, Germany, and many men in this country studied the sinuses and all expressed the belief that the lining of the sinuses should never be removed. That was my teaching for many years—in fact, until a short time ago. I have always been skeptical about the formation of a membrane on a bone—especially in a cavity the size of the antrum—that will really cover it. In reopening an antrum as large as one usually finds, which had been previously operated on and in which the entire membrane had been removed, I have found it completely covered by a new membrane—I do not say healthy mucous membrane or even mucous membrane. Most of it has the appearance of a membrane that is moist; the greater portion of it is smooth and undoubtedly functions approximately the same as the original membrane. I think there is a difference between the frontal sinus and the antrum and do not believe the new lining can be compared to that of the bony cavity of the ear after radical mastoidectomy, as here there is an entirely different epithelium. The epithelium in the ear reforms as a scar, and it is poorly nourished; there is constant desquamation.

Jansen has shown that after he experimentally grafted skin into an antrum there was constant desquamation within the cavity. This does not take place in either the frontal sinus or the antrum if it is allowed to heal naturally, as I have seen both of these after the membrane had been entirely removed. I have not seen true glands in the preparations shown here tonight or in others that I have studied. I have always felt that there were glands in the region of the ostia in these reformed membranes, but they are pseudoglands which may secrete and are in the form of folds. A number of years ago there was a report in the German literature by Tondorf, who had the courage to operate on the antrum and remove the entire membrane, using the greatest of care not to enter the nose. He did not remove any of the bone, merely the entire lining of the antrum. After packing the cavity with gauze for forty-eight hours, he reopened the antrum and removed some of the reformed membrane, which he examined histologically. He saw the same type of membrane that appears in the relining of the antrums. In some cases, after he had removed all the membrane, he took small pieces of the underlying bone and sectioned them for microscopic examination. He found tissue in the crevices of the bone, which he could not identify as epithelium but which was epithelioid. He felt that these small nests might be the cause of this regeneration of the membrane. I have not seen any confirmatory work along that line. This work, as shown by Dr. Glatt tonight, must go on until more and more is learned about the subject. The specimens shown tonight were nothing but granulation tissue covered by a single layer of flattened epithelium. The tinted preparations showed especially that they were made up mostly of granulation tissue, with a great many new blood vessels, and the supposed glands, to me, were nothing more

than cystic cavities that looked something like pseudoglands. They are lined with epithelium, but a poor kind of epithelium. Dr. Glatt's work will stimulate others to go on with this type of research.

DR. FRANK JOHN NOVAK JR.: I wish to express my admiration for the type of work shown in this paper. Dr. Beck has covered a good many of the points I had hoped to discuss; so I shall omit much that I should like to talk about. The essayist presents a view of the management of chronic disease of the maxillary sinus which is at wide variance with the accepted views at the present time. It has long been felt that the destruction, either by disease or surgically, of the lining of the maxillary sinus finished it. There was no regeneration. Then observations were made which indicated that the mucous membrane did in some measure regenerate. Then McGregor's study of 22 cases, I think, was convincing evidence that the sinus mucosa may regenerate in the regular way after total and complete destruction. Now the study of these specimens points out that while the membrane may regenerate and usually does, it does not regenerate completely. Although the epithelium lining the cavity may be of the normal stratified columnar type, the changes in the mucosa under the lining are not normal. In all these specimens there is an enormous increase in both cellular and fibrous elements of tissue. In the older specimens, taken after considerable time, the granular tissue has changed in appearance until it has become a pure fibrotic mass with relatively few vessels; in other words, it is poorly supplied with blood. There are structures in the tunica propria which look like glands; they are not normal. They have a cystic quality. On the basis of such study as this, the view that regeneration is complete and results in restoration to normal must be changed. Dr. Glatt's idea is to destroy the lining of the sinus and then forget about it—to prevent regeneration. Instead of establishing drainage to keep the ostium open, he is interested in blocking the ostium completely, and not providing accessory openings, so that the antrum will be filled with fibrous tissue or new bone, and that is what happened in cases in which his method was followed. In cases in which extensive fibrosis took place there was a clinically normal antrum.

Perhaps I have in mind a purely theoretic consideration, but it is important here: When the sinus is opened externally through the cheek, the membrane is either removed entirely—meaning complete curettement down to the bone—or only partially. If it is removed partially, there will be such things as the formation of cysts. If it is hoped that the membrane will be replaced with a tough fibrous skin, it is necessary to depend on the formation of new bone. If the entire lining is removed and complete denudation produced, the only possibility of the laying down of new bone has been removed. Bone has no capacity within itself to grow, except in the embryo. In the adult bone, if the entire periosteum is removed how can generation of new bone be possible? I think the report must be modified to this extent: The complete tunica propria must not be removed. That adjacent layer of cells which has the capacity to change to osteoblasts and thus provide for the formation of new bone must be allowed to remain. I feel kindly toward the whole proposition. It seems to me to be logical, and it seems to me that the effort to obliterate a sinus is something desirable. While it is not possible to come to a conclusion on the basis of a few slides and 15 cases, I think the work reported is a move in the right direction and should not only be carried on but be fostered elsewhere, so that a comparison of results will be available.

DR. MORRIS A. GLATT: My statement about the healing of the cavity of the middle ear after a radical mastoidectomy and the repair of skin after a total loss was not meant to be construed to indicate that these processes were analogous to the process in the sinuses after a radical operation. My aim was to question the statements that are being made that the mucosa and the glands regenerate and are histologically normal, because one thereby implies that this reformed epithelium assumes an embryonic function.

In the sinuses one is dealing with rigid walls, and since their collapse is disfiguring and dangerous, one must attempt to obliterate them with bone and scar tissue. I brought out the fact that the complete denudation of the mucosa without

ventilation will help to bring this about. One should avoid curetting the bone, because one destroys with it the bone-forming cells that adhere to the bare walls. I take exception to the curettage of the mucosa in intranasal operations, because pieces of mucosa are left in the sinuses. By the external approach it is wrong to leave pieces of mucosa because they do not appear to be diseased or to desist from entering a recess by mistaking that area for a dehiscence in the bony walls.

I can see the value of ventilation in acute, in subacute and sometimes in chronic sinusitis but not when the mucosa has been completely removed. The large "windows" or "doors" that are made in such instances for drainage are of little value, if not detrimental to the postoperative repair.

Sewal, on the other hand, deplored the obliterative process which takes place and advocated following the regular Caldwell-Luc procedure on the maxillary sinus and establishing a permanent opening in the canine fossa. He admitted that such an opening is a nuisance to the patient but stated that the end-results of leaving a thin mucosa and little obliteration of the cavity justify this as a routine procedure.

There are therefore now two opposite practices: obliteration and nonobliteration. I believe that time will prove that complete obliteration is the most effective. As to the obliteration of the ostium of a sinus, I do not attempt it, but occasionally it will become obliterated. In the frontal sinus, where the ostium is surrounded by bone, it is easily accomplished. In the maxillary sinus, where the ostium is in the membranous portion, it does not usually become obliterated. This was shown by the epithelization which has occurred in those instances in which a counter-opening was not made into the nose.

NASAL IONIZATION: HISTOLOGIC STUDIES IN RELATION TO CLINICAL EVALUATION. Presented by DR. A. R. HOLLENDER and DR. NOAH D. FABRICANT (by invitation).

This paper will be published in full in a subsequent issue of the ARCHIVES.

DISCUSSION

DR. FRANCIS L. LEDERER: Frequently a therapeutic method which posseses some merit becomes popular because with it relief of symptoms can be afforded a certain group of patients who have gone through the gamut of specific and nonspecific remedies. (This is precisely what has happened with ionization, for which, however, it must be said that Drs. Hollender and Fabricant have never claimed curative results in nasal allergy.) Then there follows a piece of work by a certain group of laboratory workers and clinicians, and whatever enthusiasm has been gained or attracted is immediately lost, because harmful mucosal effects are shown to take place. The work, naturally, is accepted as conclusive but must be reconsidered in the light of a study such as has been presented by Hollender and Fabricant. If their contentions are correct, Dean's deductions are fallacious, and a reconsideration of the ionization procedure and an evaluation of its merits purely along clinical lines, as pointed out by the essayists, are necessary. In my opinion this study is an important one, for even Dean agrees that in a certain percentage of cases nasal allergy not amenable to other measures can be relieved by ionization. One should not be too hasty in dispensing with a method until the evidence against it is conclusive.

This statement is applicable to other procedures as well as to ionization, and in this respect it emphasizes a phase of laboratory investigation which has not been properly considered in the past.

This paper then presents to me two valuable points: (1) that ionization must be judged solely on clinical results and (2) that histologic observations made on nasal mucosa after other procedures are open to question unless there is adequate histologic evidence. The single biopsy control is misleading.

DR. LEOPOLD BENNO BERNHEIMER: The essayist has spoken of evaluating ionization clinically. About a year ago I published a study, along that line, and I should like to recall the results of it.

Twenty-five patients with hyperesthetic rhinitis and 10 with seasonal hay fever were treated by zinc ionization and observed over a period of fifteen months. To compare the end-results of this treatment with those obtained with the use of such escharotics as phenol, trichloroacetic acid and zinc sulfate, 20 other patients with hyperesthetic rhinitis and 10 others with hay fever were treated with these chemicals.

By histologic observation the effects of ionization on the nasal mucous membrane were found not to differ from those of escharotics. In neither instance was there marked evidence of fibrosis, the subepithelial stroma showing little tendency to thickening after resolution was complete. With both methods there were immediate changes in surface epithelium, varying from complete destruction to simple inflammatory processes.

Clinically the course of hyperesthetic rhinitis was influenced in about 5 per cent of the patients treated with ionization, while slightly better results were obtained through the use of escharotics. The clinical course of hay fever was not altered by the use of either ionization or escharotics.

The most important conclusion drawn, however, was that ionization carries a definite risk of complications that do not present themselves through the use of escharotics. In 2 cases in which ionization was used anosmia developed. This persisted for four months in 1 case, and for seven months in the other. In 1 case there developed unilateral headache, typical of the syndrome discussed by Sluder. This persisted for three and one-half months. The majority of patients treated by ionization complained of headache, sometimes associated with nausea, lasting for many hours after ionization. I have never encountered a complication following the use of escharotics.

My belief is that, as the relief of symptoms in allergic nasal disease is less striking through the use of ionization than with escharotics and since ionization carries risks of complications not encountered with escharotics, it is time the ionizing machine is dropped from the armamentarium of the rhinologist.

Dr. Frank John Novak Jr.: There are several points that should be kept in mind. In the first place, what is normal epithelium or normal mucosa? It can be seen only in the new-born infant who was not exposed to syphilis in the mother or to other conditions. If he is normal, the mucosa of the nose is normal. There is no adult in this climate who has not had changes in the nose. These changes are more or less constant and definite, and practitioners know what they are. Therefore, it is fairly reasonable to accept the view that in an adult in this climate, exposed to the things to which he is known to be exposed, the mucosa can be labeled normal for that person under those conditions. The changes in the epithelium will vary constantly. The stratified squamous epithelium is known to be present in the anterior part of the septum. The lateral walls and the septum are known to consist of normal pseudostratified epithelium. If a biopsy specimen is taken from the anterior part of the nose in 1 case and from the middle turbinate in another case, the 2 cases cannot be compared. In a series in which biopsy specimens are always taken from the same place, I think it can be said that the histologic picture represents normal. There are changes which vary from normal. There is thickening in the membrana propria. This is known to be composed, partly at least, of a certain type of fibers which take silver. When these fibers are separated by edema, the membrana propria presents the appearance of thickening. There are changes, too, in the submucosa, in the tunica propria. If these tissues were stained or impregnated with silver, one could make a more or less quantitative estimation of changes in the tissue of both the membrana propria and the tunica propria. Mention was made of the various types of epithelium in the nose. There are only three types, the pseudostratified, the cuboidal and the pavement type of epithelium. There is no transitional type.

I am firmly convinced that, while ionization may be deleterious to the nasal mucosa, curettage, which is done so regularly, produces much more damage.

Dr. Samuel Salinger: I concede that Dr. Hollender is one of the outstanding proponents of ionization as a therapeutic agent and has had a vast experience

which merits respect. However, it seems to me that, far from strengthening his stand on this proposition, his presentation this evening has greatly weakened his position, and he has definitely taken a step backward when he states that the evaluation of the procedure will have to be made on clinical grounds. I had hoped and had even been led to believe that there would be forthcoming evidence based on histologic studies which would prove that ionization was not only a beneficial agent but a safe one. This evening's presentation fails to offer any substantial refutation of Dean's and McMahon's work as to the effects of ionization on the mucous membrane. As a matter of fact, Dr. Hollender admits that changes do take place but offers the alibi that similar changes are found in tissues that have never been exposed to ionization. The important facts which all the members want to know are: How much damage does ionization do? Is the damage permanent? Do the results justify the trauma inflicted? I hope that the authors will not be discouraged but will continue their studies in an effort to answer these highly important questions.

DR. J. A. WEISS (by invitation): Has the essayist any information as to whether the ionized nasal mucous membrane ever becomes approximately normal in function by physiologic standards, such as the absorption of active substances, for instance the secretion of the pituitary gland? If so, how long after ionization may that be expected?

DR. NOAH D. FABRICANT (by invitation): I wish to thank the discussers for their comments on the paper. In regard to the complications that follow ionization—anosmia, headache, nausea and vomiting—which Dr. Bernheimer has observed in his experience, I can say that in all our work at the University of Illinois we have not encountered them. I myself have not observed any case in which anosmia followed ionization at our clinic. We use the strip gauze method, and we do not pack the nose too high. Possibly such complications occur, and perhaps it has been only our good luck that we have not had them. I can imagine that through faulty technic such things might happen. Whether escharotics or ionization is best is not a problem that comes up under the title of our paper. I know escharotics have proved of value, but I feel that ionization has a place in the therapeutic armament. We ask the same trial for ionization as is given patients after an allergic survey. It has been mentioned that escharotics should be prescribed and ionization dropped, but I think it can be admitted that escharotics have their failures. Ionization has had a degree of success in various hands, as well as of failure.

Dr. Novak suggested that in making biopsies the specimen should be taken from the same mathematical point each time. I feel that we covered that point and illustrated that weakness, for the reason that if a biopsy specimen is taken from a certain area before ionization and another is taken afterward, there will be trauma before ionization and trauma after ionization. There is resultant scar tissue. Then if a specimen is taken from the same site there will be trauma piled on trauma, and I am afraid the picture will be misleading.

The question was asked whether studies in physiologic stains have been made. They have not been, and I think that is a problem for future work that merits consideration. I want to make one remark from L. W. Dean (The Fundamentals of Allergic Rhinitis with Particular Reference to Ionization, *Ann. Otol., Rhin. & Laryng.* **45**:326 [June] 1936). Dean is more optimistic than I am. He makes the statement that ionization does improve allergic rhinitis in most cases. I feel it may improve it in many but not most. McMahon made this statement in a general discussion on allergic rhinitis. "There should be some place in our therapeutic armamentarium for a method for which such beneficial clinical results are claimed." If the changes in the tissues are apt to cause complications, let practitioners not employ ionization; if they are not, let us give the method a trial. I do not feel that one should say they are dangerous and then use the method. If the changes cause only partial disability, I should say use of the method is feasible.

Book Reviews

The Rehabilitation of Speech: A Textbook of Diagnostic and Corrective Procedures. By Robert West, Ph.D., Professor of Speech Pathology, University of Wisconsin; Lou Kennedy, Ph.D., Associate Professor of Speech, Brooklyn College, New York, and Anna Carr, M.A., Clinical Adviser in Speech, State Teachers College, Milwaukee, Wisconsin. Price, $4. Pp. 475, with 28 illustrations. New York: Harper & Brothers, 1937.

In the preface the authors tell how difficult it is to write a book such as this. In spite of the fact that the subject of speech disorders and their rehabilitation is unlike most specialized subjects and that no one person could be well informed in all branches of study contributory to the rehabilitation of speech, the authors are to be congratulated for presenting a really fine textbook.

They have compressed in 475 pages a tremendous amount of information, arranged in a logical and coherent manner. All phases of the subject have been covered. This alone makes the textbook of vast practical importance.

Knowledge of speech rehabilitation has accumulated rapidly in recent years, to the end that some standardization of opinion has been achieved and definite principles have been promulgated. The authors have incorporated the universally accepted doctrines, setting them forth in the order of their importance, and have at all times excluded controversial matters, even if the latter may seem to have been successfully proved. For this they are to be commended.

Throughout the text, one notes with what thoroughness various phases of speech disorders are discussed. It is evident that the authors have gone to great lengths to be as accurate and as precise as possible. Such terms as "speech disorders" and "speech defects" have been sharply differentiated. Throughout, one notes that a workable classification has been preferred. The entire theme has been grouped under four etiologic categories: physiologic, anatomic, psychogenic and imitative. Though the text deals with the first three chiefly, many exercises are found that will aid one in dealing with the "imitative" conditions.

Part 1 deals with diagnostic procedures. The neurophysiologic mechanism for speech is discussed in moderate detail, the endocrine, autonomic and central nervous system being considered and compared. Minute attention is paid to the various functions of these systems. Then, in logical order, follows a discussion of the normal versus the abnormal. All phases of possible psychogenic disorders are briefly but comprehensively covered. The chapter on stuttering (dysphemia) is highly instructive. The authors have preferred to incorporate in this chapter what the prevailing point of view of the English writers seems to be, namely, to use the word stuttering rather than stammering for the type of disorder described. After each chapter, either therapy or prognosis is moderately discussed. Speech disorders arising from deformity of the mouth, nasal obstruction, pharyngeal or laryngeal deformity or a neuropathologic condition are adequately discussed, with illustrations of anatomic studies and photographs. Every effort has been made to include any condition or group of conditions that might cause a speech disorder (dyslalia). Several pages are devoted to the speech defects of the feebleminded, and a much larger number are assigned to the very important dyslalia due to deficiency of hearing. The details of the more important aspects of various dyslalias are given proper attention. The dyslalias of speech perception, especially the principles of case study and diagnosis, are so thoroughly and minutely discussed that it is impossible to even begin to describe the immense value of this chapter to the speech clinician.

Part 2 embraces the remedial procedures for older children and adults. It describes details of the speech clinic and general exercises for relaxation (for the lips, the tongue and the velum, for breathing and for voice control) and covers in a comprehensive manner the various commonly encountered special articulatory defects of the consonants. Remedial measures for indistinct speech and all measures

for training and retraining to overcome the disorders of speech fully discussed in part 1 follow in logical order. The general plan of remedial measures is carried out in part 3, under the title "Remedial Measures for Children." Part 4 deals with the influence of foreign language, and part 5, with speech reading. Some case histories are given in appendix A, whereas in appendix B some very fine technic is given for the examination of the physical organs of speech. Here again, as in many other portions of this fine work, one is impressed with precise detail and with the clearcut instruction. Appendix C covers adequately the various methods of testing hearing acuity. In appendix D are described tests of articulation, and appendix E includes height and weight tables for children from 5 to 18 years of age. Appendix F contains instructions for calculating the index of dental maturation, and appendix G includes the high points of the training of elementary school teachers for speech correction.

A glossary of 35 pages is included for the purpose of increasing the usability of the book. A complete bibliography appears at the end of the book, in order to aid the reader to consult collateral works. The index is complete.

The entire step-up of the book, including the good type, and the excellent reading will impress the sincere student.

In connection with the treatment of laryngeal and pharyngeal stenoses, the authors make the interesting remark that "the tonsils are not needed for good voice." In their removal, however, the authors state that if the faucial pillars are cut or cicatrices are left which constrict the faucial opening, the voice may be permanently affected. Should the patient fail to follow the surgeon's instructions for postoperative care or fail to exercise the throat freely while the wounds are healing, in order to prevent adhesions which would affect the movements of the palatopharyngeal and palatoglossal muscles, then the patient takes on his own head the responsibility of later vocal difficulties (pages 99 and 100).

In conclusion, this book contains so much material that it will be useful not only to the speech clinician but to the physician interested in disorders and defects of speech, to the medical student, to the dentist and the oral surgeon, to the otolaryngologist and, lastly, to the graduate student in otolaryngology and oral surgery.

Diseases of the Nose, Throat and Ear. By I. Simson Hall, M.B., Ch.B., F.R.C.P.E., F.R.C.S.E., Edinburgh. Price, $4. Pp. 407, with 391 illustrations, including one plate in color. Baltimore: William Wood & Company, 1937.

This volume is, as the author has designated, purely a handbook for the medical student and the general practitioner. The work is concise, has good arrangement of material, is well written and contains the essentials necessary for instruction of the practitioner in diseases of the nose, throat and ear. However, it is much too lacking in detail to be suitable for the postgraduate student or for one who wishes more than the elementary essentials. It contains many practical suggestions for the treatment of the common diseases in the field concerned, but its usefulness could have been enhanced by more illustrations, particularly in the sections on diseases of the throat and of the ear. In some parts of the treatise opinions are expressed with which some otolaryngologists as experienced as the author will find some differences. In the appendix are instructions for the preparation of patients for various operative procedures and a number of prescriptions recommended by the author for the treatment of some of the diseases mentioned.

Diseases of the Ear, Throat and Nose. By J. Douglas McLaggan, M.A., M.B., F.R.C.S. England and Edinburgh; Surgeon in Charge of the Ear, Nose and Throat Department, Royal Free Hospital; Surgeon, Central London Throat, Nose and Ear Hospital; Surgeon in Charge of the Ear, Nose and Throat Department, Croydon General Hospital. Price, 15 shillings. Pp. 338. London: H. K. Lewis & Co., Ltd., 1937.

This book is well written. It covers systematically the most important principles of otolaryngologic work. The final chapter is devoted to "Allergic Diseases in Relation to the Nose," which subject is covered in six pages.

The author approaches the subject from the anatomic side, for which he is to be commended. His indications for operative procedure are clearly set forth. The value of this text might have been enhanced if the author had added a list of drugs and medicaments as well as prescriptions to the chapters on treatment.

Description of certain technics and procedures is given due emphasis. The author's explanation of the "Technique of Eustachian Catheterization" and the method of "Passage of the Catheter," as outlined on pages 124 to 127, deserves special praise.

Though this book is a small one, it is none the less thorough. It should be of great help to the medical student and to the busy general practitioner. It is doubtful how valuable it would be to the graduate student in otolaryngology, to the resident in this field or to the specialist.

News and Comment

THIRD ANNUAL POSTGRADUATE COURSE IN OPHTHALMOLOGY AND OTOLARYNGOLOGY

The third annual postgraduate course in ophthalmology and otolaryngology will be given jointly by the Oregon Academy of Ophthalmology and Otolaryngology and the University of Oregon Medical School, April 3 to 9, 1938. Drs. A. C. Furstenberg and Sanford Gifford will be the guest teachers. Anatomic and surgical demonstrations will be given at the University of Oregon Medical School. Dr. Paul Bailey, Medical-Dental Building, Portland, is secretary of the course.

Directory of Otolaryngologic Societies *

FOREIGN

Collegium Oto-Rhino-Laryngologicum Amicitiæ Sacrum

President: Prof. H. Burger, Keizersgracht 317 C, Amsterdam.
Secretary: Prof. Dr. C. E. Benjamins, Verlengde Heereweg 143, Groningen, Netherlands.

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Gordon B. New, Mayo Clinic, Rochester, Minn.
Secretary: Dr. Leroy A. Schall, 270 Commonwealth Ave., Boston.
Place: San Francisco. Time: June 13-17, 1938.

American Academy of Ophthalmology and Otolaryngology

President: Dr. Harry S. Gradle, 58 E. Washington St., Chicago.
Executive Secretary: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.

American Bronchoscopic Society

President: Dr. Gordon Berry, 36 Pleasant St., Worcester, Mass.
Secretary: Dr. Lyman Richards, 319 Longwood Ave., Boston.
Place: Atlantic City, N. J.

American Laryngological Association

President: Dr. J. F. Barnhill, Miami Beach, Fla.
Secretary: Dr. James A. Babbitt, 1912 Spruce St., Philadelphia.
Place: Atlantic City, N. J.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. Samuel J. Kopetzky, 71 E. 80th St., New York.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester, N. Y.
Place: Atlantic City, N. J. Time: April 27-29, 1938.

SECTIONS:

Eastern.—Chairman: Dr. Louis H. Clerf, 1530 Locust St., Philadelphia.
Place: Philadelphia. Time: Jan. 7, 1938.
Southern.—Chairman: Dr. Murdock S. Equen, Medical Arts Bldg., Atlanta, Ga.
Place: Atlanta, Ga. Time: Jan. 24, 1938.
Middle.—Chairman: Dr. James B. Costen, Beaumont Bldg., St. Louis.
Place: St. Louis, Mo. Time: Jan. 26, 1938.
Western.—Chairman: Dr. Arthur C. Jones, 105 N. 8th St., Boise, Idaho.
Place: Santa Barbara, Calif. Time: Jan. 30-31, 1938.

American Otological Society

President: Dr. Harris P. Mosher, 828 Beacon St., Boston.
Secretary: Dr. Thomas J. Harris, 104 E. 40th St., New York.
Place: Atlantic City, N. J.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.